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* * * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * * * *
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=> FILE BIOSIS, CABA, CAPLUS, EMBASE, JAPIO, LIFESCI, MEDLINE, SCISEARCH, USPATFULL
=> e garssen jan gerrit/au
E1            3     GARSSEN J E/AU
E2            1     GARSSEN JAN E/AU
E3            0 --> GARSSEN JAN GERRIT/AU
E4          114     GARSSEN JOHAN/AU
E5            2     GARSSEN M P/AU
E6           12     GARSSEN M P J/AU
E7            5     GARSSEN MARCEL P J/AU
E8            1     GARSSHOFF UTA/AU
E9            6     GARSSON B/AU
E10           1     GARSSON BARRY/AU
E11           2     GARSSON HENRY M/AU
E12           2     GARSSON R M/AU

=>  e garssen jan/au
E1            1     GARSSEN J */AU
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E3            0 --> GARSSEN JAN/AU
E4            1     GARSSEN JAN E/AU
E5          114     GARSSEN JOHAN/AU
E6            2     GARSSEN M P/AU
E7           12     GARSSEN M P J/AU
E8            5     GARSSEN MARCEL P J/AU
E9            1     GARSSHOFF UTA/AU
E10           6     GARSSON B/AU
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E12           2     GARSSON HENRY M/AU

=> e jacobs jorg gunther/au
E1            1     JACOBS JORG/AU
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E4            2     JACOBS JORN/AU
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E7            1     JACOBS JOSE C/AU
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E11           2     JACOBS JOSEPH ANTHONY/AU
E12           9     JACOBS JOSEPH B/AU

=> s e1-e3
L1            3 ("JACOBS JORG"/AU OR "JACOBS JORG GUENTHER"/AU OR "JACOBS JORG
                GUNTHER"/AU)

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2             3 DUP REM L1 (0 DUPLICATES REMOVED)

=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L2   ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2004 ACS on STN
AN   2004:57406  CAPLUS
DN   140:124865
TI   Detection of prion disease
IN   Van Oers, Josephus Wilhelmus Alphonsus Maria; Hack, Cornelis Erik;
     Roem-Haagsma, Dorina Dirkje Ina; Langeveld, Johannes Pieter Maria;
     Garssen, Gerrit Jan;   ***Jacobs, Jorg Guenther***  ; Van Engelenburg,
     Franciscus Antonius Cornelis
PA   Pepscan Systems B.V., Neth.; Stichting Sanquin Bloedvoorziening
SO   Eur. Pat. Appl., 38 pp.
     CODEN: EPXXDW
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
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PI	EP 1382971	A1	20040121	EP 2002-77910	20020717
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
	WO 2004007555	A2	20040122	WO 2003-NL523	20030717
	WO 2004007555	A3	20040401		
	W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
PRAI	EP 2002-77910	A	20020717		

AB This invention relates to the field of the detection of prion diseases. The invention provides a binding mol. or antibody specifically reactive with an epitope which is exposed on a part of an aberrant conformer (PrPSc) of a prion protein after treatment of said conformer with a protease wherein said epitope is not or only partly exposed on a prion protein which has not been treated with a protease.

RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 2 OF 3 USPATFULL on STN
AN 2003:208289 USPATFULL
TI Methods and apparatus for determination and decrease of dynamic positioning errors of an ablating laser during refractive laser surgery
IN Teiwes, Winfried, Teltow/Berlin, GERMANY, FEDERAL REPUBLIC OF
Huppertz, Michael, Teltow/Berlin, GERMANY, FEDERAL REPUBLIC OF
Weise, Ralf, Teltow/Berlin, GERMANY, FEDERAL REPUBLIC OF
Jacobs, Jorg , Teltow/Berlin, GERMANY, FEDERAL REPUBLIC OF

PI	US 2003144651	A1	20030731
AI	US 2002-276768	A1	20021119 (10)
	WO 2001-EP5837		20010521

DT Utility
FS APPLICATION
LREP THE FIRM OF KARL F ROSS, 5676 RIVERDALE AVENUE, PO BOX 900, RIVERDALE (BRONX), NY, 10471-0900
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 8 Drawing Page(s)
LN.CNT 388
AB The above described apparatus and methods provide the possibility to reduce or even eliminate the effects of delay between image acquisition and laser ablation. Thus this will lead to less positioning errors and therefore to better ablation results in laser refractive surgery. The importance of this invention will increase with decreasing ablating beam diameter. The use of synchronization leads to shorter delay times. Hence it follows that the duration of the whole treatment decreases as well.

L2 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:574024 CAPLUS
DN 133:174276
TI Prion test using guanidine thiocyanate for reducing false positive test results
IN Garssen, Gerrit Jan; ***Jacobs, Jorg Gunther*** ; Langeveld, Joannes Pieter Maria; Smits, Marinus Adrianus; Van Keulen, Lucien Johannes Mattheus; Schreuder, Bram Edward Cornelis; Bossers, Alexander
PA Stichting Dienst Landbouwkundig Onderzoek, Neth.
SO PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI WO 2000048003	A1	20000817	WO 2000-NL79	20000209

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             CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL,
             IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA,
             MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI,
             SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM,
             AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
             DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
             CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     EP 1151305           A1    20011107    EP 2000-904139          20000209
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO
PRAI EP 1999-200391       A     19990211
     WO 2000-NL79         W     20000209
AB   The invention is related to diagnostic methods for detecting transmissible
     spongiform encephalopathies (TSEs) such as BSE and scrapie and related
     disease in humans.  The invention provides use of guanidine thiocyanate
     (gdnSCN) or a functional equiv. thereof for treating at least one sample
     derived from a mammal, including humans for reducing the risk of scoring a
     false-pos. test result in testing said sample for the presence or absence
     of aberrant prion protein.
RE.CNT  4     THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
              ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> e garssen gerrit jan/au
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E2            2     GARSSEN GERRIT J/AU
E3            2 --> GARSSEN GERRIT JAN/AU
E4            1     GARSSEN HANS GEORG/AU
E5            3     GARSSEN HOEKSTRA J/AU
E6          379     GARSSEN J/AU
E7            1     GARSSEN J */AU
E8            3     GARSSEN J E/AU
E9            1     GARSSEN JAN E/AU
E10         114     GARSSEN JOHAN/AU
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=> s e2-e3
L3            4 ("GARSSEN GERRIT J"/AU OR "GARSSEN GERRIT JAN"/AU)

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4             4 DUP REM L3 (0 DUPLICATES REMOVED)

=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y


L4   ANSWER 1 OF 4  CAPLUS  COPYRIGHT 2004 ACS on STN
AN   2004:57406  CAPLUS
DN   140:124865
TI   Detection of prion disease
IN   Van Oers, Josephus Wilhelmus Alphonsus Maria; Hack, Cornelis Erik;
     Roem-Haagsma, Dorina Dirkje Ina; Langeveld, Johannes Pieter Maria;
       ***Garssen, Gerrit Jan***  ; Jacobs, Jorg Guenther; Van Engelenburg,
     Franciscus Antonius Cornelis
PA   Pepscan Systems B.V., Neth.; Stichting Sanquin Bloedvoorziening
SO   Eur. Pat. Appl., 38 pp.
     CODEN: EPXXDW
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
     ----------          ----   --------    -------------------    --------
PI   EP 1382971           A1    20040121    EP 2002-77910          20020717
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK
     WO 2004007555         A2    20040122    WO 2003-NL523          20030717
     WO 2004007555         A3    20040401
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W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI EP 2002-77910 A 20020717

AB This invention relates to the field of the detection of prion diseases. The invention provides a binding mol. or antibody specifically reactive with an epitope which is exposed on a part of an aberrant conformer (PrPSc) of a prion protein after treatment of said conformer with a protease wherein said epitope is not or only partly exposed on a prion protein which has not been treated with a protease.

RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN

AN 2000:574024 CAPLUS

DN 133:174276

TI Prion test using guanidine thiocyanate for reducing false positive test results

IN ***Garssen, Gerrit Jan*** ; Jacobs, Jorg Gunther; Langeveld, Joannes Pieter Maria; Smits, Marinus Adrianus; Van Keulen, Lucien Johannes Mattheus; Schreuder, Bram Edward Cornelis; Bossers, Alexander

PA Stichting Dienst Landbouwkundig Onderzoek, Neth.

SO PCT Int. Appl., 49 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000048003	A1	20000817	WO 2000-NL79	20000209
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
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EP 1151305	A1	20011107	EP 2000-904139	20000209
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRAI EP 1999-200391 A 19990211
WO 2000-NL79 W 20000209

AB The invention is related to diagnostic methods for detecting transmissible spongiform encephalopathies (TSEs) such as BSE and scrapie and related disease in humans. The invention provides use of guanidine thiocyanate (gdnSCN) or a functional equiv. thereof for treating at least one sample derived from a mammal, including humans for reducing the risk of scoring a false-pos. test result in testing said sample for the presence or absence of aberrant prion protein.

RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN

AN 1978:46572 CAPLUS

DN 88:46572

TI Studies on DNA unwinding. Proton and phosphorus nuclear magnetic resonance studies of gene V protein from bacteriophage M13, interacting with d(pC-G-C-G)

AU ***Garssen, Gerrit J.*** ; Hilbers, Cornelis W.; Schoenmakers, John G. G.; Van Boom, Jacques H.

CS Lab. Biofys. Chem., Univ. Nijmegen, Nijmegen, Neth.

SO European Journal of Biochemistry (1977), 81(3), 453-63
CODEN: EJBCAI; ISSN: 0014-2956
DT Journal
LA English
AB The interaction of gene V protein from bacteriophage M13 with the self-complementary tetranucleotide d(pC-G-C-G) was studied by 1H and 31P NMR. Using the H-bonded proton resonances of the Watson-Crick base pairs as a probe, it is shown that the protein is able to unwind the small double-helical fragment even at 0.degree.. Binding of the tetranucleotide causes changes in the arom. part of the 1H NMR spectrum of the complex, suggesting that arom. residues, most likely tyrosines, take part in the protein-nucleic acid interaction. From the 31P NMR spectra of the protein-nucleic acid complex, it follows that the pK value of the 5'-terminal phosphate is lower than for the free nucleic acid species. Moreover, the exchange of protein between nucleic acid substrates is fast. Combination of these measurements suggests a mechanism of unwinding on the tetranucleotide level. To a large extent the unwinding is detd. by fluctuations in the double-helical DNA structure.

L4 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1976:101428 CAPLUS
DN 84:101428
TI The formation of threo-11-hydroxy-trans-12:13-epoxy-9-cis-octadecenoic acid by enzymic isomerization of 13-L-hydroperoxy-9-cis,11-trans-octadecadienoic acid by soybean lipoxygenase-1
AU ***Garssen, Gerrit J.*** ; Veldink, Gerrit A.; Vliegenthart, Johannes F. G.; Boldingh, Jan
CS Org. Chem. Lab., Rijksuniv., Utrecht, Neth.
SO European Journal of Biochemistry (1976), 62(1), 33-6
CODEN: EJBCAI; ISSN: 0014-2956
DT Journal
LA English
AB The interaction of soybean lipoxygenase-1 with 13-L-hydroperoxy-9-cis,11-trans-octadecadienoic acid (13-hydroperoxylinoleate), the product of the enzymic dioxygenation of linoleic acid, gave either a yellow or a purple-colored enzyme species depending on the amt. of product used. With an excess of 13-hydroperoxylinoleate, a labile purple-colored enzyme species was formed which reverted to a yellow-colored form with concomitant conversion of the hydroperoxy compd. In this reaction, 13-hydroperoxylinoleate isomerized into threo-11-hydroxy-trans-12:13-epoxy-9-cis-octadecenoic acid as could be concluded from NMR and mass spectral data. Expts. with hydroperoxylinoleate-13-18O2 showed a high retention (70%) of the hydroperoxy O atoms in the end product.

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=> e langeveld joannes pierter/au
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E7           2     LANGEVELD JOHANNES PIETER MARIA/AU
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E9           1     LANGEVELD KLERKS ADRIANA HENDRIKA/AU
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E12          3     LANGEVELD L/AU

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L5          15 ("LANGEVELD JAN P"/AU OR "LANGEVELD JAN P M"/AU OR "LANGEVELD
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               PRION?

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6            6 DUP REM L5 (9 DUPLICATES REMOVED)

=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 6 ANSWERS - CONTINUE? Y/(N):y
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L6 ANSWER 1 OF 6 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 1
AN 2004:77483 BIOSIS
DN PREV200400079379
TI Enzymatic degradation of ***prion*** protein in brain stem from infected cattle and sheep.
AU ***Langeveld, Jan P. M.*** [Reprint Author]; Wang, Jeng-Jie; van de Wiel, Dick F. M.; Shih, Giles C.; Garssen, G. Jan; Bossers, Alex; Shih, Jason C. H.
CS Central Institute for Animal Disease Control, 8203 AA, PO Box 2004, Lelystad, Netherlands
jan.langeveld@wur.nl
SO Journal of Infectious Diseases, (1 December 2003) Vol. 188, No. 11, pp. 1782-1789. print.
CODEN: JIDIAQ. ISSN: 0022-1899.
DT Article
LA English
ED Entered STN: 4 Feb 2004
Last Updated on STN: 4 Feb 2004
AB ***Prions*** -infectious agents involved in transmissible spongiform encephalopathies-normally survive proteolytic and mild protein-destructive processes. Using bacterial keratinase produced by Bacillus licheniformis strain PWD-1, we tested conditions to accomplish the full degradation of ***prion*** protein (PrP) in brain-stem tissue from animals with bovine spongiform encephalopathy and scrapie. The detection of PrPSc, the disease-associated isoform of PrP, in homogenates was done by Western blotting and various antibodies. The results indicated that only in the presence of detergents did heat pretreatment at >100degreeC allow the extensive enzymatic breakdown of PrPSc to a state where it is immunochemically undetectable. Proteinase K and 2 other subtilisin proteases, but not trypsin and pepsin, were also effective. This enzymatic process could lead to the development of a method for the decontamination of medical and laboratory equipment. The ultimate effectiveness of this method of ***prion*** inactivation has to be tested in mouse bioassays.

L6 ANSWER 2 OF 6 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 2
AN 2002:572409 BIOSIS
DN PREV200200572409
TI PrPCWD lymphoid cell targets in early and advanced chronic wasting disease of mule deer.
AU Sigurdson, Christina J.; Barillas-Mury, Carolina; Miller, Michael W.; Oesch, Bruno; Van Keulen, Lucien J. M.; ***Langeveld, Jan P. M.*** ; Hoover, Edward A. [Reprint author]
CS Department of Microbiology, Immunology and Pathology, College of Veterinary, Colorado State University, Fort Collins, CO, 80523-1671, USA
ehoover@lamar.colostate.edu
SO Journal of General Virology, (October, 2002) Vol. 83, No. 10, pp. 2617-2628. print.
CODEN: JGVIAY. ISSN: 0022-1317.
DT Article
LA English
ED Entered STN: 7 Nov 2002
Last Updated on STN: 7 Nov 2002
AB Up to 15% of free-ranging mule deer in northeastern Colorado and southeastern Wyoming, USA, are afflicted with a ***prion*** disease, or transmissible spongiform encephalopathy (TSE), known as chronic wasting disease (CWD). CWD is similar to a subset of TSEs including scrapie and variant Creutzfeldt-Jakob disease in which the abnormal ***prion*** protein isoform, PrPCWD, accumulates in lymphoid tissue. Experimental scrapie studies have indicated that this early lymphoid phase is an important constituent of ***prion*** replication interposed between mucosal entry and central nervous system accumulation. To identify the lymphoid target cells associated with PrPCWD, we used triple-label immunofluorescence and high-resolution confocal microscopy on tonsils from naturally infected deer in advanced disease. We detected PrPCWD primarily extracellularly in association with follicular dendritic and B cell membranes as determined by frequent co-localization with antibodies against membrane bound immunoglobulin and CD21. There was minimal co-localization with cytoplasmic labels for follicular dendritic cells

(FDC). This finding could indicate FDC capture of PrPCWD, potentially in association with immunoglobulin or complement, or PrPC conversion on FDC. In addition, scattered tingible body macrophages in the germinal centre contained coarse intracytoplasmic aggregates of PrPCWD, reflecting either phagocytosis of PrPCWD on FDC processes, apoptotic FDC or B cells, or actual PrPCWD replication within tingible body macrophages. To compare lymphoid cell targets in early and advanced disease, we also examined: (i) PrPCWD distribution in lymphoid cells of fawns within 3 months of oral CWD exposure and (ii) tonsil biopsies from preclinical deer with naturally acquired CWD. These studies revealed that the early lymphoid cellular distribution of PrPCWD was similar to that in advanced disease, i.e. in a pattern suggesting FDC association. We conclude that in deer, PrPCWD accumulates primarily extracellularly and associated with FDCs and possibly B cells - a finding which raises questions as to the cells responsible for pathological ***prion*** production.

L6 ANSWER 3 OF 6 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 3
AN 2002:574352 BIOSIS
DN PREV200200574352
TI Temporal and spatial relationship between the death of PrP-damaged neurones and microglial activation.
AU Bate, Clive [Reprint author]; Boshuizen, Ronald S.; ***Langeveld, Jan P.*** *** M.*** ; Williams, Alun
CS Department of Veterinary Pathology, Veterinary School, Institute of Comparative Medicine, University of Glasgow, Bearsden Road, Glasgow, G61 1QH, UK
SO Neuroreport, (16 September, 2002) Vol. 13, No. 13, pp. 1695-1700. print.
CODEN: NERPEZ. ISSN: 0959-4965.
DT Article
LA English
ED Entered STN: 7 Nov 2002
Last Updated on STN: 7 Nov 2002
AB Previous studies have demonstrated a role for microglia in the neuronal loss that occurs in the transmissible spongiform encephalopathies or ***prion*** diseases. In the present studies, the processes that lead to the death of neurones treated with synthetic peptides derived from the ***prion*** protein (PrP) were fully activated within 1 h, although neuronal cell death was not seen until 24 h later. Similarly, neurones exposed to PrP peptides for only 1 h activated microglia and a temporal relationship between the production of interleukin-6, an indicator of microglial activation, and microglial killing of PrP-treated neurones was also demonstrated. Activation of microglia and microglia-mediated killing of PrP-treated neurones or scrapie-infected neuroblastoma cells were maximal only when microglia were in direct contact with neurones.

L6 ANSWER 4 OF 6 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 4
AN 2002:172961 BIOSIS
DN PREV200200172961
TI Adult human microglia secrete cytokines when exposed to neurotoxic ***prion*** protein peptide: No intermediary role for prostaglandin E2.
AU Veerhuis, Robert [Reprint author]; Hoozemans, Jeroen J. M.; Janssen, Ingrid; Boshuizen, Ronald S.; ***Langeveld, Jan P. M.*** ; Eikelenboom, Piet
CS Department of Psychiatry, Research Institute Neurosciences Vrije Universiteit, Vrije Universiteit Medical Center, Amsterdam, Netherlands r.veerhuis@vumc.nl
SO Brain Research, (25 January, 2002) Vol. 925, No. 2, pp. 195-203. print.
CODEN: BRREAP. ISSN: 0006-8993.
DT Article
LA English
ED Entered STN: 5 Mar 2002
Last Updated on STN: 5 Mar 2002
AB ***Prion*** diseases are characterized by accumulation of protease resistant isoforms of ***prion*** protein (termed PrPSC), glial activation and neurodegeneration. The time course of PrP deposition, appearance of activated microglia, and of neuronal apoptosis in experimentally-induced ***prion*** disease suggests that microglial activation precedes the process of neuronal loss. Activated microglia and inflammatory mediators, including cytokines and prostaglandin E2 (PGE2)

co-localize with PrP deposits. In vitro, mouse microglia secrete neurotoxic agents and interleukins (IL)-1 and IL-6, when exposed to synthetic peptides representing the neurotoxic fragment of PrP. In this study, adult human microglia were found to secrete IL-6 and TNF-alpha upon exposure to synthetic fibrillar PrP105-132, the putative transmembrane domain of PrP. Little cytokine release occurred following exposure of microglia to C-terminally amidated, nonfibrillar PrP105-132, suggesting that the degree of fibrillarity of PrP peptides affects their biological properties. Non-steroidal anti-inflammatory drugs (NSAIDs) are thought to exert beneficial effects in neurodegenerative disorders through suppressive effects on microglial activation and on cyclooxygenase (COX) activity. Since microglial COX-2 expression and PGE2 synthesis are increased in human and experimental ***prion*** diseases, we investigated the effects of the NSAIDs indomethacin and BF389, an experimental COX-2 selective inhibitor, on the PrP105-132-induced microglial IL-6 and TNF-alpha synthesis in vitro. No inhibitory effects of the NSAIDs were observed. Furthermore, PrP105-132 did not stimulate microglial PGE2 synthesis. We conclude that, unlike IL-1beta-induced IL-6 synthesis in astrocytes, the PrP-induced IL-6 synthesis in human adult microglia is not PGE2 mediated.

L6 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:574024 CAPLUS
DN 133:174276
TI ***Prion*** test using guanidine thiocyanate for reducing false positive test results
IN Garssen, Gerrit Jan; Jacobs, Jorg Gunther; ***Langeveld, Joannes Pieter*** *** Maria*** ; Smits, Marinus Adrianus; Van Keulen, Lucien Johannes Mattheus; Schreuder, Bram Edward Cornelis; Bossers, Alexander
PA Stichting Dienst Landbouwkundig Onderzoek, Neth.
SO PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000048003	A1	20000817	WO 2000-NL79	20000209
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1151305	A1	20011107	EP 2000-904139	20000209
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
PRAI EP 1999-200391	A	19990211		
WO 2000-NL79	W	20000209		

AB The invention is related to diagnostic methods for detecting transmissible spongiform encephalopathies (TSEs) such as BSE and scrapie and related disease in humans. The invention provides use of guanidine thiocyanate (gdnSCN) or a functional equiv. thereof for treating at least one sample derived from a mammal, including humans for reducing the risk of scoring a false-pos. test result in testing said sample for the presence or absence of aberrant ***prion*** protein.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 6 OF 6 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 5
AN 1998:322054 BIOSIS
DN PREV199800322054
TI An antibody raised against a conserved sequence of the ***prion*** protein recognizes pathological isoforms in human and animal ***prion*** diseases, including Creutzfeldt-Jakob disease and bovine spongiform encephalopathy.
AU Piccardo, Pedro [Reprint author]; ***Langeveld, Jan P. M.*** ; Hill,

Andrew F.; Dlouhy, Stephen R.; Young, Katherine; Giaccone, Giorgio; Rossi, Giacomina; Bugiani, Marianna; Bugiani, Orso; Meloen, Rob H.; Collinge, John; Tagliavini, Fabrizio; Ghetti, Bernardino

CS Indiana Univ. Med. Center, Div. Neuropathology, 635 Barnhill Drive, MS A-142, Indianapolis, IN, USA

SO American Journal of Pathology, (June, 1998) Vol. 152, No. 6, pp. 1415-1420. print.
CODEN: AJPAA4. ISSN: 0002-9440.

DT Article

LA English

ED Entered STN: 22 Jul 1998
Last Updated on STN: 22 Jul 1998

AB Antibodies to the ***prion*** protein (PrP) have been critical to the neuropathological and biochemical characterization of PrP-related degenerative diseases in humans and animals. Although PrP is highly con served evolutionarily, there is some sequence divergence among species; as a consequence, anti-PrP antibodies have a wide spectrum of reactivity (from strong immunopositivity to lack of reactivity) when challenged with PrP from diverse species. We have produced an antibody (anti-PrP95-108) raised against a synthetic peptide corresponding to residues 95 to 108 of human PrP and have characterized it by epitope mapping, Western immunoblot analysis, and immunohistochemistry. The antibody recognizes not only human PrP isoforms but also pathological PrP from all species tested (i.e., cattle, sheep, hamsters, and mice). This is probably due to the fact that the epitope recognized by this antibody includes residues 100 to 108 of human PrP, a sequence that is also present in PrP of several other species. Thus, this reagent is valuable not only for the study of human ***prion*** diseases but also for analysis of the possible relationship between human and animal disorders.

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=> smits marinus a/au
SMITS IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> e smits marinus a/au
E1            1     SMITS MARIANNE E/AU
E2            1     SMITS MARIEKE T/AU
E3            1 --> SMITS MARINUS A/AU
E4            1     SMITS MARINUS A L/AU
E5            7     SMITS MARINUS ADRIANUS/AU
E6            7     SMITS MARIUS/AU
E7            1     SMITS MARK/AU
E8            2     SMITS MARK M/AU
E9            7     SMITS MARTIJNTJE/AU
E10           2     SMITS MARTIN/AU
E11           2     SMITS MARTINO M/AU
E12           2     SMITS MATTHIJS/AU

=> s e3-e5
L7            9 ("SMITS MARINUS A"/AU OR "SMITS MARINUS A L"/AU OR "SMITS MARINU
                S ADRIANUS"/AU)

=> dup rem l7
PROCESSING COMPLETED FOR L7
L8             7 DUP REM L7 (2 DUPLICATES REMOVED)

=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 7 ANSWERS - CONTINUE? Y/(N):y

L8   ANSWER 1 OF 7  CAPLUS  COPYRIGHT 2004 ACS on STN
AN   2003:791415  CAPLUS
DN   139:287271
TI   Methods and kits for detection of single nucleotide polymorphisms in
     nucleic acids
IN   Agbo, Edwin Chukwura; Te Pas, Marinus Frederick Willem;   ***Smits,***
  ***     Marinus Adrianus***
PA   Id-Lelystad, Instituut voor Dierhouderij en Diergezondheid B. V., Neth.
SO   Eur. Pat. Appl., 28 pp.
```

CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1350853	A1	20031008	EP 2002-76336	20020405
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
WO 2003087409	A1	20031023	WO 2003-NL253	20030404
W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
PRAI EP 2002-76336	A	20020405		

AB The present invention provides methods for producing a nucleic acid fingerprint as well as methods for detecting sequence polymorphisms between one or more genomes. The methods involve fragmenting a nucleic acid into nucleic acid fragments with ends that are compatible to ligation with at least one adapter, performing a ligation reaction between the compatible ends of the nucleic acid fragments and at least one adapter, amplifying the nucleic acid fragments by using at least one amplification primer, and generating a nucleic acid fingerprint from the amplified fragments. A method according to the present invention permits high-resoln. fingerprinting while maintaining stringency in a PCR reaction. In another aspect, the invention also provides a kit for prepg. nucleic acid fingerprints according to methods of the invention.

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 2 OF 7 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 1
AN 2003:85902 BIOSIS
DN PREV200300085902
TI Recombinant vaccine for prevention and/or treatment of pleuropneumonia infections.
AU Kamp, Elbarte Margriet [Inventor, Reprint Author]; ***Smits, Marinus*** *** Adrianus*** [Inventor]
CS Wijngaard 27, 8212 CC Lelystad, Netherlands
PI US 6500435 December 31, 2002
SO Official Gazette of the United States Patent and Trademark Office Patents, (Dec 31 2002) Vol. 1265, No. 5. http://www.uspto.gov/web/menu/patdata.html . e-file.
ISSN: 0098-1133 (ISSN print).
DT Patent
LA English
ED Entered STN: 6 Feb 2003
Last Updated on STN: 6 Feb 2003

AB The invention provides a vaccine for the prevention and/or the treatment of infection by Actinobacillus pleuropneumoniae, the causative agent of porcine pleuropneumonia, which vaccine contains at least an immunogenic part of at least one cytolytic protein of A. pleuropneumoniae produced by recombinant DNA, and detoxified derivatives thereof. Three of such cytolytic proteins are identified and a vaccine containing these, or parts or derivatives thereof, ensures protection against all known serotypes of A. pleuropneumoniae. The cytolytic proteins are produced by inserting a nucleotide sequence encoding one or more of the proteins or parts thereof in a host cell, cultivating the host cell and recovering the proteins. Another vaccine contains the genetic information for one or more of the cytolytic proteins, and a passive vaccine contains antibodies against these proteins. The invention further provides monoclonal antibodies and DNA probes for use in diagnostics.

L8 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2004 ACS on STN

AN 2000:574024 CAPLUS
DN 133:174276
TI Prion test using guanidine thiocyanate for reducing false positive test results
IN Garssen, Gerrit Jan; Jacobs, Jorg Gunther; Langeveld, Joannes Pieter Maria; ***Smits, Marinus Adrianus*** ; Van Keulen, Lucien Johannes Mattheus; Schreuder, Bram Edward Cornelis; Bossers, Alexander
PA Stichting Dienst Landbouwkundig Onderzoek, Neth.
SO PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000048003	A1	20000817	WO 2000-NL79	20000209
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1151305	A1	20011107	EP 2000-904139	20000209
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
PRAI EP 1999-200391	A	19990211		
WO 2000-NL79	W	20000209		

AB The invention is related to diagnostic methods for detecting transmissible spongiform encephalopathies (TSEs) such as BSE and scrapie and related disease in humans. The invention provides use of guanidine thiocyanate (gdnSCN) or a functional equiv. thereof for treating at least one sample derived from a mammal, including humans for reducing the risk of scoring a false-pos. test result in testing said sample for the presence or absence of aberrant prion protein.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 4 OF 7 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 2
AN 2000:277831 BIOSIS
DN PREV200000277831
TI Nucleic acid encoding Actinobacillus pleuropneumoniae cytolytic proteins.
AU Kamp, Elbarte Margriet [Inventor, Reprint author]; ***Smits, Marinus*** *** Adrianus*** [Inventor]
CS Wijngaard 27, 8212 CC Lelystad, Netherlands
PI US 5994525 November 30, 1999
SO Official Gazette of the United States Patent and Trademark Office Patents, (Nov. 30, 1999) Vol. 1228, No. 5. e-file.
CODEN: OGUPE7. ISSN: 0098-1133.
DT Patent
LA English
ED Entered STN: 6 Jul 2000
Last Updated on STN: 7 Jan 2002
AB The invention provides a vaccine for the prevention and/or the treatment of infection by Actinobacillus pleuropneumoniae, the causative agent of porcine pleuropneumonia, which vaccine contains at least an immunogenic part of at least one cytolytic protein of A. pleuropneumoniae produced by recombinant DNA, and detoxified derivatives thereof. Three of such cytolytic proteins are identified and a vaccine containing these, or parts or derivatives thereof, ensures protection against all known serotypes of A. pleuropneumoniae. The cytolytic proteins are produced by inserting a nucleotide sequence encoding one or more of the proteins or parts thereof in a host cell, cultivating the host cell and recovering the proteins. another vaccine contains the genetic information for one or more of the cytolytic proteins, and a passive vaccine contains antibodies against these proteins. The invention further provides monoclonal antibodies and DNA probes for use in diagnostics.

L8 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1997:679268 CAPLUS
DN 127:316560
TI Method for the detection of prion diseases
IN Schreuder, Bram Edward Cornelis; Van Keulen, Lucius Johannes Mattheus; Vromans, Maria Elisabeth Wilhelmina; Langeveld, Johannes Pieter Maria; ***Smits, Marinus Adrianus***
PA Instituut voor Dierhouderij en Diergezondheid (Id-Dlo), Neth.; Schreuder, Bram Edward Cornelis; Van Keulen, Lucius Johannes Mattheus; Vromans, Maria Elisabeth Wilhelmina; Langeveld, Johannes Pieter Maria; Smits, Marinus Adrianus
SO PCT Int. Appl., 29 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9737227	A1	19971009	WO 1997-NL166	19970402
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	CA 2250800	AA	19971009	CA 1997-2250800	19970402
	AU 9721808	A1	19971022	AU 1997-21808	19970402
	AU 713529	B2	19991202		
	EP 891552	A1	19990120	EP 1997-914658	19970402
	EP 891552	B1	20030402		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	BR 9708421	A	19990803	BR 1997-8421	19970402
	NZ 332132	A	20000228	NZ 1997-332132	19970402
	JP 2000505559	T2	20000509	JP 1997-535157	19970402
	JP 3333213	B2	20021015		
	AT 236407	E	20030415	AT 1997-914658	19970402
	NO 9804602	A	19981203	NO 1998-4602	19981001
PRAI	EP 1996-200917	A	19960403		
	WO 1997-NL166	W	19970402		

AB The invention provides methods for the detection of prion diseases, such as scrapie of sheep, bovine spongiform encephalopathy of cattle, Creutzfeld-Jacob disease of man, whereby aberrant proteins or prion proteins are detected in tissues which can be sampled from live animals. Peptides such as segments of the scrapie protein can be used to raise antibodies for use in immunoassays of lymphoid tissues such as the tonsils.

L8 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1993:624252 CAPLUS
DN 119:224252
TI Recombinant vaccine for prevention and/or treatment of pleuropneumonia infections
IN Kamp, Elbarte M.; ***Smits, Marinus A.***
PA Centraal Diergeneeskundig Instituut, Neth.
SO Can. Pat. Appl., 50 pp.
CODEN: CPXXEB
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	CA 2045950	AA	19921229	CA 1991-2045950	19910628
	US 5994525	A	19991130	US 1995-488706	19950609
	US 6500435	B1	20021231	US 1998-62126	19980417
PRAI	CA 1991-2045950	A	19910628		
	US 1991-722971	B1	19910628		
	US 1993-138609	B3	19931015		
	US 1995-488706	A1	19950609		

```
AB   A vaccine is provided for the prevention and/or treatment of infection by
     Actinobacillus pleuropneumoniae, the causative agent of porcine
     pleuropneumonia.  The vaccine contains at least an immunogenic part of
     .gtoreq.1 cytolytic protein of A. pleuropneumoniae produced by recombinant
     DNA, and detoxified derivs. thereof.  Three such cytolytic proteins
     (cytolysins I and II and III) are identified, and a vaccine contg. these,
     or parts or derivs. thereof, ensures protection against all known
     serotypes of A. pleuropneumoniae.  Another vaccine contains the genetic
     information for .gtoreq.1 of the cytolytic proteins, and a passive vaccine
     contains antibodies against these proteins.  Also disclosed are monoclonal
     antibodies (MAbs) and DNA probes for use in diagnostics.  Gene cloning and
     identification of the cytolysins are described, as are heterogeneity in
     the cytolysin II genetic determinant of A. pleuropneumoniae serotypes,
     identification of hemolytic and cytotoxic proteins of A. pleuropneumoniae
     by MAbs, prodn. of cytolysins, and prepn. of a recombinant vaccine.

L8   ANSWER 7 OF 7  USPATFULL on STN
AN     83:3620  USPATFULL
TI     Scaffolding system
IN       ***Smits, Marinus A. L.***  , Henley-on-Klip, South Africa
PA     SGB Group Limited, Surrey, England (non-U.S. corporation)
PI     US 4369859              19830125
AI     US 1981-225385          19810115 (6)
PRAI   ZA 1980-225         19800115
DT     Utility
FS     Granted
EXNAM  Primary Examiner: Machado, Reinaldo P.
LREP   Buell, Blenko, Ziesenheim & Beck
CLMN   Number of Claims: 10
ECL    Exemplary Claim: 1
DRWN   4 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 283
AB     In a scaffolding system an upright tube is provided with an annular dish
       shaped protrusion. A housing on the end of a horizontal tube carries a
       lever with a cam face that engages the protrusion to lock the tubes
       together. The horizontal tube can be at any radial orientation relative
       to the upright tube.


=> e van keulen lucien/au
E1          45     VAN KEULEN L J M/AU
E2           1     VAN KEULEN LISA/AU
E3           2 --> VAN KEULEN LUCIEN/AU
E4           3     VAN KEULEN LUCIEN J M/AU
E5           1     VAN KEULEN LUCIEN JOHANNES MATTHEUS/AU
E6           1     VAN KEULEN LUCIUS JOHANNES MATTHEUS/AU
E7          16     VAN KEULEN M/AU
E8           7     VAN KEULEN M A/AU
E9           2     VAN KEULEN MARRIT/AU
E10          1     VAN KEULEN MICHAEL/AU
E11          4     VAN KEULEN MIKE/AU
E12          1     VAN KEULEN NICK/AU

=> s e1-e6 and prion?
L9          46 ("VAN KEULEN L J M"/AU OR "VAN KEULEN LISA"/AU OR "VAN KEULEN
               LUCIEN"/AU OR "VAN KEULEN LUCIEN J M"/AU OR "VAN KEULEN LUCIEN
               JOHANNES MATTHEUS"/AU OR "VAN KEULEN LUCIUS JOHANNES MATTHEUS"/A
               U) AND PRION?

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10          21 DUP REM L9 (25 DUPLICATES REMOVED)

=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 21 ANSWERS - CONTINUE? Y/(N):y

L10  ANSWER 1 OF 21  CAPLUS  COPYRIGHT 2004 ACS on STN DUPLICATE 1
AN   2004:299720  CAPLUS
DN   141:5358
TI   Discrimination between scrapie and bovine spongiform encephalopathy in
     sheep by molecular size, immunoreactivity, and glycoprofile of
```

prion protein

AU Thuring, C. M. A.; Erkens, J. H. F.; Jacobs, J. G.; Bossers, A.; ***Van*** *** Keulen, L. J. M.*** ; Garssen, G. J.; Van Zijderveld, F. G.; Ryder, S. J.; Groschup, M. H.; Sweeney, T.; Langeveld, J. P. M.

CS Central Institute for Animal Disease Control, Lelystad, 8203 AA, Neth.

SO Journal of Clinical Microbiology (2004), 42(3), 972-980
CODEN: JCMIDW; ISSN: 0095-1137

PB American Society for Microbiology

DT Journal

LA English

AB A procedure for discrimination between scrapie and bovine spongiform encephalopathy (BSE) in sheep is of importance for establishing whether BSE has entered the sheep population. Since BSE has not yet been found in sheep at the farm level, such discrimination procedures can be developed only with exptl. sheep BSE. Two distinctive mol. features of the ***prion*** protein (PrP)-mol. size and glycosylation profile-in proteinase K digests of brain stem tissue from sheep were used here; upon Western blotting, these features led to an unequivocal discrimination among natural scrapie, exptl. scrapie, and exptl. BSE. The higher electrophoretic mobility of PrP in sheep BSE could be best obsd. after deglycosylation treatment with N-glycosidase F. A simpler method for confirmation of this size difference involved comparison of the ratios for the binding of two monoclonal antibodies: P4 and 66.94b4. Based on epitope mapping studies with P4 and peptides, it appeared that N-terminal amino acid sequence WGQGGSH was intact only in sheep scrapie digests. Another feature typical for PrP in sheep BSE was the large fraction of diglycosylated PrP (70% or more). These data were obtained for a large group of pos. sheep, consisting of 7 sheep with exptl. BSE infection (genotypes: 6 ARQ/ARQ and one AHQ/AHQ), 48 sheep naturally infected with scrapie (6 different genotypes), and 3 sheep with primary exptl. scrapie infection. Routine tests of slaughter material serve well for the initial detection of both BSE and scrapie. With Western blotting as a rapid follow-up test, a 66.94b4/P4 antibody binding ratio above 1.5 is a practical indicator for serious suspicion of BSE infection in sheep.

RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 2 OF 21 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 2

AN 2002:572409 BIOSIS

DN PREV200200572409

TI PrPCWD lymphoid cell targets in early and advanced chronic wasting disease of mule deer.

AU Sigurdson, Christina J.; Barillas-Mury, Carolina; Miller, Michael W.; Oesch, Bruno; ***Van Keulen, Lucien J. M.*** ; Langeveld, Jan P. M.; Hoover, Edward A. [Reprint author]

CS Department of Microbiology, Immunology and Pathology, College of Veterinary, Colorado State University, Fort Collins, CO, 80523-1671, USA
ehoover@lamar.colostate.edu

SO Journal of General Virology, (October, 2002) Vol. 83, No. 10, pp. 2617-2628. print.
CODEN: JGVIAY. ISSN: 0022-1317.

DT Article

LA English

ED Entered STN: 7 Nov 2002
Last Updated on STN: 7 Nov 2002

AB Up to 15% of free-ranging mule deer in northeastern Colorado and southeastern Wyoming, USA, are afflicted with a ***prion*** disease, or transmissible spongiform encephalopathy (TSE), known as chronic wasting disease (CWD). CWD is similar to a subset of TSEs including scrapie and variant Creutzfeldt-Jakob disease in which the abnormal ***prion*** protein isoform, PrPCWD, accumulates in lymphoid tissue. Experimental scrapie studies have indicated that this early lymphoid phase is an important constituent of ***prion*** replication interposed between mucosal entry and central nervous system accumulation. To identify the lymphoid target cells associated with PrPCWD, we used triple-label immunofluorescence and high-resolution confocal microscopy on tonsils from naturally infected deer in advanced disease. We detected PrPCWD primarily extracellularly in association with follicular dendritic and B cell membranes as determined by frequent co-localization with antibodies against membrane bound immunoglobulin and CD21. There was minimal

co-localization with cytoplasmic labels for follicular dendritic cells (FDC). This finding could indicate FDC capture of PrPCWD, potentially in association with immunoglobulin or complement, or PrPC conversion on FDC. In addition, scattered tingible body macrophages in the germinal centre contained coarse intracytoplasmic aggregates of PrPCWD, reflecting either phagocytosis of PrPCWD on FDC processes, apoptotic FDC or B cells, or actual PrPCWD replication within tingible body macrophages. To compare lymphoid cell targets in early and advanced disease, we also examined: (i) PrPCWD distribution in lymphoid cells of fawns within 3 months of oral CWD exposure and (ii) tonsil biopsies from preclinical deer with naturally acquired CWD. These studies revealed that the early lymphoid cellular distribution of PrPCWD was similar to that in advanced disease, i.e. in a pattern suggesting FDC association. We conclude that in deer, PrPCWD accumulates primarily extracellularly and associated with FDCs and possibly B cells - a finding which raises questions as to the cells responsible for pathological ***prion*** production.

L10 ANSWER 3 OF 21 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 3
AN 2002:284421 BIOSIS
DN PREV200200284421
TI Early and late pathogenesis of natural scrapie infection in sheep.
AU ***van Keulen, L. J. M.*** [Reprint author]; Vromans, M. E. W.; van Zijderveld, F. G.
CS Institute for Animal Science and Health (ID-Lelystad), NL-8200 AB, Lelystad, Netherlands
L.J.M.vanKeulen@id.wag-ur.nl
SO APMIS, (January, 2002) Vol. 110, No. 1, pp. 23-32. print.
CODEN: APMSEL. ISSN: 0903-4641.
DT Article
LA English
ED Entered STN: 8 May 2002
Last Updated on STN: 8 May 2002
AB The pathogenesis of scrapie infection was studied in sheep carrying the PrPVRQ/PrPVRQ genotype, which is associated with a high susceptibility for natural scrapie. The sheep were killed at sequential time points during a scrapie infection covering both the early and late stages of scrapie pathogenesis. Various lymphoid and neural tissues were collected and immunohistochemically examined for the presence of the scrapie-associated ***prion*** protein PrPSc, a marker forscrapie infectivity. The first stage of scrapie infection consisted of invasion of the palatine tonsil and Peyer's patches of the caudal jejunum and ileum, the so-called gut-associated lymphoid tissues (GALT). At the same time, PrPSc was detected in the medial retropharyngeal lymph nodes draining the palatine tonsil and the mesenteric lymph nodes draining the jejunal and ileal Peyer's patches. From these initial sites of scrapie replication, the scrapie agent disseminated to other non-GALT-related lymphoid tissues. Neuroinvasion started in the enteric nervous system followed by retrograde spread of the scrapie agent via efferent parasympathetic and sympathetic nerve fibres innervating the gut, to the dorsal motor nucleus of the vagus in the medulla oblongata and the intermediolateral column of the thoracic spinal cord segments T8-T10, respectively.

L10 ANSWER 4 OF 21 CABA COPYRIGHT 2004 CABI on STN
AN 2001:79586 CABA
DN 20013069876
TI Inactivation of ***prions*** by rendering processes
Nachhaltige Tierproduktion
AU Schreuder, B. E. C.; Geertsma, R. E.; Keulen, L. J. M. van; Enthoven, P.; Oberthur, R. C.; Koeijer, A. A. de; Osterhaus, A. D. M. E.; ***van*** *** Keulen, L. J. M.*** ; de Koeijer, A. A.; Kamphues, J. [EDITOR]; Flachowsky, G. [EDITOR]
CS Institute for Animal Science and Health (ID-Lelystad), P.O.Box 65, 8200 AB, Lelystad, Netherlands.
SO Landbauforschung Volkenrode, Sonderheft, (2001) No. 223, pp. 130-141. 23 ref.
Publisher: Bundesforschungsanstalt fur Landwirtschaft (FAL). Braunschweig
Price: Journal article; Conference paper .
Meeting Info.: Animal nutrition - resources and future developments. Workshop on Sustainable Animal Production, 15-16 June 2000, EXPO 2000, Hannover, Germany.

ISSN: 0376-0723; ISBN: 3-933140-47-1
CY Germany, Federal Republic of
DT Journal
LA English
SL German
ED Entered STN: 20010802
Last Updated on STN: 20010802
AB The present study assesses the efficacy of the procedures in use at rendering plants working with a hyperbaric system. The experiments were performed on a laboratory-scale using procedures simulating the pressure cooking part of the rendering procedures. As spike materials, a pool of BSE infected brain stem material from the UK and one of scrapie infected brain stem materials from Dutch sheep were used to spike rendering materials. These mixtures were subjected to various time-temperature combinations of hyperbaric heat treatment related to the Dutch rendering conditions in the early nineties, as well as to the combination of 20 minutes at 133 [deg]C indicated in the EU Directive on rendering of 1996. With the 20 min 133 [deg]C procedure, a reduction of BSE infectivity was observed of about 2.2 log in the first round (with some residual infectivity detected), and in the second round in excess of 2.0 log (no residual infectivity detected). Data obtained with undiluted brain material indicated an inactivation, in this form, of about 3.0 log (with some residual infectivity detected). With the same procedure, scrapie infectivity was reduced by more than 1.7 log in the first series and more than 2.2 log in the second series. Results with undiluted brain material indicated an inactivation, in this form, in excess of 3.1 log. In all three cases with the scrapie material, no residual infectivity was detected. Especially in processes with lower time-temperature exposure, the BSE agent consistently appeared more resistant to heat inactivation procedures than the scrapie agent.

L10 ANSWER 5 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:574024 CAPLUS
DN 133:174276
TI ***Prion*** test using guanidine thiocyanate for reducing false positive test results
IN Garssen, Gerrit Jan; Jacobs, Jorg Gunther; Langeveld, Joannes Pieter Maria; Smits, Marinus Adrianus; ***Van Keulen, Lucien Johannes*** *** Mattheus*** ; Schreuder, Bram Edward Cornelis; Bossers, Alexander
PA Stichting Dienst Landbouwkundig Onderzoek, Neth.
SO PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000048003	A1	20000817	WO 2000-NL79	20000209
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1151305	A1	20011107	EP 2000-904139	20000209
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
PRAI EP 1999-200391	A	19990211		
WO 2000-NL79	W	20000209		

AB The invention is related to diagnostic methods for detecting transmissible spongiform encephalopathies (TSEs) such as BSE and scrapie and related disease in humans. The invention provides use of guanidine thiocyanate (gdnSCN) or a functional equiv. thereof for treating at least one sample derived from a mammal, including humans for reducing the risk of scoring a false-pos. test result in testing said sample for the presence or absence of aberrant ***prion*** protein.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 6 OF 21 CABA COPYRIGHT 2004 CABI on STN
AN 2001:90149 CABA
DN 20013068534
TI Pathogenesis of natural scrapie in sheep
Archives of Virology, Supplement 16
AU Keulen, L. J. M. van; Schreuder, B. E. C.; Vromans, M. E. W.; Langeveld, J. P. M.; Smits, M. A.; ***van Keulen, L. J. M.*** ; Groschup, M. H. [EDITOR]; Kretzschmar, H. [EDITOR]
CS Institute for Animal Science and Health (ID-Lelystad), Edelhertweg 15, PO Box 65, NL-8200 AB Lelystad, Netherlands.
SO Prion diseases: diagnosis and pathogenesis, (2000) pp. 57-71. 33 ref.
Publisher: Springer-Verlag Wien. Wien
ISBN: 3-211-83530-X
CY Austria
DT Book; Book Article
LA English
ED Entered STN: 20010906
Last Updated on STN: 20010906
AB Although scrapie has been known for a long time as a natural disease of sheep and goats, the pathogenesis in its natural host still remains unclear. To study the pathogenesis of natural scrapie, we used immunohistochemistry to monitor the deposition of PrPSc in various tissues, collected during a natural scrapie infection from sheep with the PrPVRQ/PrPVRQ genotype which were purposely bred for their short incubation period for natural scrapie. PrPSc was present in the lymphoid tissues of all animals from the age of 5 months onwards. At this age, PrPSc was detected in the neural tissues only in the enteric nervous system (ENS) at the level of the duodenum and ileum. At the age of 10 months, PrPSc was not only found in the ENS but also in the ganglion mesentericum cranialis/coeliacum, the dorsal motor nucleus of the vagus, and the intermediolateral column of the thoracic segments T8-T10. PrPSc was detected for the first time in the nucleus tractus solitarius and ganglion nodosus at 17 months of age and in the ganglion trigeminale and several spinal ganglia at 21 months of age. Since the scrapie agent consists largely, if not entirely of PrPSc, these results indicate that the ENS acts as a portal of entry to the neural tissues for the scrapie agent followed by centripetal and retrograde spread through sympathetic and parasympathetic efferent fibres of the autonomic nervous system to the spinal cord and medulla oblongata respectively. PrPSc accumulation in sensory ganglia occurs after infection of the CNS and is therefore probably due to centrifugal and anterograde spread of the scrapie agent from the CNS through afferent nerve fibres.

L10 ANSWER 7 OF 21 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 4
AN 2001:430765 BIOSIS
DN PREV200100430765
TI Early accumulation of PrPSc in gut-associated lymphoid and nervous tissues of susceptible sheep from a Romanov flock with natural scrapie.
AU Andreoletti, Olivier; Berthon, Patricia [Reprint author]; Marc, Daniel; Sarradin, Pierre; Grosclaude, Jeanne; ***van Keulen, Lucien*** ; Schelcher, Francois; Elsen, Jean-Michel; Lantier, Frederic
CS Laboratoire de Pathologie Infectieuse et Immunologie, INRA, F-37380, Nouzilly, France
berthon@tours.inra.fr
SO Journal of General Virology, (December, 2000) Vol. 81, No. 12, pp. 3115-3126. print.
CODEN: JGVIAY. ISSN: 0022-1317.
DT Article
LA English
ED Entered STN: 12 Sep 2001
Last Updated on STN: 22 Feb 2002
AB The immune system is known to be involved in the early phase of scrapie pathogenesis. However, the infection route of naturally occurring scrapie and its spread within the host are not entirely known. In this study, the pathogenesis of scrapie was investigated in sheep of three PrP genotypes, from 2 to 9 months of age, which were born and raised together in a naturally scrapie-affected Romanov flock. The kinetics of PrPSc accumulation in sheep organs were determined by immunohistochemistry. PrPSc was detected only in susceptible VRQ/VRQ sheep, from 2 months of

age, with an apparent entry site at the ileal Peyer's patch as well as its draining mesenteric lymph node. At the cellular level, PrPSc deposits were associated with CD68-positive cells of the dome area and B follicles before being detected in follicular dendritic cells. In 3- to 6-month-old sheep, PrPSc was detected in most of the gut-associated lymphoid tissues (GALT) and to a lesser extent in more systemic lymphoid formations such as the spleen or the mediastinal lymph node. All secondary lymphoid organs showed a similar intensity of PrPSc-immunolabelling at 9 months of age. At this time-point, PrPSc was also detected in the autonomic myenteric nervous plexus and in the nucleus parasympathicus nervi X of the brain stem. These data suggest that natural scrapie infection occurs by the oral route via infection of the Peyer's patches followed by replication in the GALT. It may then spread to the central nervous system through the autonomic nervous fibres innervating the digestive tract.

L10 ANSWER 8 OF 21 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 5
AN 2001:16897 BIOSIS
DN PREV200100016897
TI Diagnosis of bovine spongiform encephalopathy: A review.
AU ***van Keulen, L. J. M.*** ; Langeveld, J. P. M.; Garssen, G. J.; Jacobs, J. G.; Schreuder, B. E. C.; Smits, M. A. [Reprint author]
CS Institute for Animal Science and Health (ID-Lelystad), Edelhertweg 15, NL-8200 AB, Lelystad, Netherlands
L.J.M.vanKeulen@id.wag-ur.nl
SO Veterinary Quarterly, (October, 2000) Vol. 22, No. 4, pp. 197-200. print.
CODEN: VEQUDU. ISSN: 0165-2176.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 27 Dec 2000
Last Updated on STN: 27 Dec 2000
AB Cows affected with bovine spongiform encephalopathy (BSE) display chronic neurological signs consisting of behavioural changes, abnormalities of posture and movement, and/or hyperaesthesia. At present, there are no laboratory test available to diagnose BSE in the live animal. In this article, we describe the post-mortem diagnostic examination of brains from BSE-suspected cattle as currently performed at ID-Lelystad. The routine laboratory diagnosis of BSE consists of histopathological examination of the brain and detection of the modified ***prion*** protein, PrPBSE, in brain tissue. These tests, however, have the disadvantage of being laborious and time consuming, so that results are available only after several days. Recently, at ID-Lelystad a new post-mortem test has been developed that enables screening of larger volumes of brain samples for PrPBSE within 1 day. This BSE test is especially suited for slaughterline monitoring. A preliminary validation study has shown that both sensitivity and specificity are 100% compared to the gold diagnostic standard of histopathology.

L10 ANSWER 9 OF 21 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 2001:129849 BIOSIS
DN PREV200100129849
TI Pathogenesis of natural scrapie in sheep.
AU ***van Keulen, L. J. M.*** [Reprint author]; Schreuder, B. E. C.; Vromans, M. E. W.; Langeveld, J. P. M.; Smits, M. A.
CS Institute for Animal Science and Health (ID-Lelystad), Edelhertweg 15, NL-8200 AB, Lelystad, Netherlands
SO Archives of Virology Supplement, (2000) No. 16, pp. 57-71. print.
CODEN: AVISE9. ISSN: 0939-1983.
DT Article
LA English
ED Entered STN: 14 Mar 2001
Last Updated on STN: 15 Feb 2002
AB Although scrapie has been known for a long time as a natural disease of sheep and goats, the pathogenesis in its natural host still remains unclear. To study the pathogenesis of natural scrapie, we used immunohistochemistry to monitor the deposition of PrPSc in various tissues, collected during a natural scrapie infection from sheep with the PrPVRQ/PrPVRQ genotype which were purposely bred for their short incubation period for natural scrapie. PrPSc was present in the lymphoid

tissues of all animals from the age of 5 months onwards. At this age, PrPSc was detected in the neural tissues only in the enteric nervous system (ENS) at the level of the duodenum and ileum. At the age of 10 months, PrPSc was not only found in the ENS but also in the ganglion mesentericum cranialis/coeliacum, the dorsal motor nucleus of the vagus, and the intermediolateral column of the thoracic segments T8-T10. PrPSc was detected for the first time in the nucleus tractus solitarius and ganglion nodosus at 17 months of age and in the ganglion trigeminale and several spinal ganglia at 21 months of age. Since the scrapie agent consists largely, if not entirely of PrPSc, these results indicate that the ENS acts as a portal of entry to the neural tissues for the scrapie agent followed by centripetal and retrograde spread through sympathetic and parasympathetic efferent fibers of the autonomic nervous system to the spinal cord and medulla oblongata respectively. PrPSc accumulation in sensory ganglia occurs after infection of the CNS and is therefore probably due to centrifugal and anterograde spread of the scrapie agent from the CNS through afferent nerve fibers.

L10 ANSWER 10 OF 21 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 2001:73376 SCISEARCH
GA The Genuine Article (R) Number: 392PR
TI Pathogenesis of natural scrapie in sheep
AU ***van Keulen L J M (Reprint)*** ; Schreuder B E C; Vromans M E W; Langeveld J P M; Smits M A
CS Inst Anim Sci & Hlth, ID Lelystad, Edelhertweg 15, POB 65, NL-8200 AB Lelystad, Netherlands (Reprint); Inst Anim Sci & Hlth, ID Lelystad, NL-8200 AB Lelystad, Netherlands
CYA Netherlands
SO ARCHIVES OF VIROLOGY, (JAN 2000) Supp. [16], pp. 57-71.
Publisher: SPRINGER-VERLAG WIEN, SACHSENPLATZ 4-6, PO BOX 89, A-1201 VIENNA, AUSTRIA.
ISSN: 0304-8608.
DT Article; Journal
LA English
REC Reference Count: 33
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Although scrapie has been known for a long time as a natural disease of sheep and goats, the pathogenesis in its natural host still remains unclear. To study the pathogenesis of natural scrapie, we used immunohistochemistry to monitor the deposition of PrPSc in various tissues, collected during a natural scrapie infection from sheep with the PrPVRQ/PrPVRQ genotype which were purposely bred for their short incubation period for natural scrapie. PrPSc was present in the lymphoid tissues of all animals from the age of 5 months onwards. At this age, PrPSc was detected in the neural tissues only in the enteric nervous system (ENS) at the level of the duodenum and ileum. At the age of 10 months, PrPSc was not only found in the ENS but also in the ganglion mesentericum cranialis/coeliacum, the dorsal motor nucleus of the vagus, and the intermediolateral column of the thoracic segments T8-T10. PrPSc was detected for the first time in the nucleus tractus solitarius and ganglion nodosus at 17 months of age and in the ganglion trigeminale and several spinal ganglia at 31 months of age. Since the scrapie agent consists largely, if not entirely of PrPSc, these results indicate that the ENS acts as a portal of entry to the neural tissues for the scrapie agent followed by centripetal and retrograde spread through sympathetic and parasympathetic efferent fibers of the autonomic nervous system to the spinal cord and medulla oblongata respectively. PrPSc accumulation in sensory ganglia occurs after infection of the CNS and is therefore probably due to centrifugal and anterograde spread of the scrapie agent from the CNS through afferent nerve fibers.

L10 ANSWER 11 OF 21 CABA COPYRIGHT 2004 CABI on STN DUPLICATE 6
AN 2001:22588 CABA
DN 20003020403
TI Applicability of three anti-Prp peptide sera including staining of tonsils and brainstem of sheep with scrapie
AU Garssen, G. J.; Keulen, L. J. M. van; Farquhar, C. F.; Smits, M. A.; Jacobs, J. G.; Bossers, A.; Meloen, R. H.; Langeveld, J. P. M.; ***van*** *** Keulen, L. J. M.***
CS Department of Molecular Recognition, Institute for Animal Science and

Health (ID-Lelystad), Lelystad, Netherlands.
SO Microscopy Research and Technique, (2000) Vol. 50, No. 1, pp. 32-39. 28 ref.
Publisher: Wiley-Liss. New York
ISSN: 1059-910x
CY United States
DT Journal
LA English
ED Entered STN: 20010302
Last Updated on STN: 20010302
AB Three rabbit antibodies (R521, R505, R524) were produced, and raised to synthetic peptides corresponding to residues 94-105, 100-111 and 223-234, respectively, of the sheep ***prion*** protein (PrP). Epitope mapping analysis revealed the monospecific character of antisera R505 and R524. In addition to the amino acid sequence against which it was raised, R521 also recognized other small epitopes. ELISA and radio-immunoprecipitation were used to assess the relative immunoreactivities of the antisera to the normal sheep ***prion*** protein (PrPc). Highest reactivity was found for R521, followed by R505 and R524. According to Western blot analysis, all three sera specifically reacted with the ***prion*** proteins PrPSc and PrP27-30, extracted from the brain stem of a scrapie-affected sheep. Yet, with R505 not all of the lower molecular weight deglycosylated forms could be detected. Contrary to the immunoreactivities found with the PrPSc and PrP27-30 isoforms, only R521 recognised PrPc from a healthy sheep. The usefulness of all three anti-peptide sera in the immunohistochemical detection of PrPSc in brain stem and tonsils of scrapie-affected sheep was demonstrated and compared with an established rabbit anti-PrP serum.

L10 ANSWER 12 OF 21 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 7
AN 1999:359362 BIOSIS
DN PREV199900359362
TI Scrapie-associated ***prion*** protein in the gastrointestinal tract of sheep with natural scrapie.
AU ***van Keulen, L. J. M.*** [Reprint author]; Schreuder, B. E. C. [Reprint author]; Vromans, M. E. W. [Reprint author]; Langeveld, J. P. M.; Smits, M. A.
CS Department of Pathobiology and Epidemiology, Institute for Animal Science and Health (ID-DLO), Edelhertweg 15, NL-8200 AB, Lelystad, Netherlands
SO Journal of Comparative Pathology, (July, 1999) Vol. 121, No. 1, pp. 55-63. print.
CODEN: JCVPAR. ISSN: 0021-9975.
DT Article
LA English
ED Entered STN: 2 Sep 1999
Last Updated on STN: 2 Sep 1999
AB The scrapie-associated ***prion*** protein (PrPSc), which is closely associated with scrapie infectivity, accumulates in the brain and lymphoid tissues of sheep with natural scrapie. The most probable portal of entry of the scrapie agent in sheep is the alimentary tract; little attention, however, has been paid to the gastro-intestinal tract in scrapie research. In this study, we examined the presence and distribution of PrPSc within the gastro-intestinal tract of sheep with natural scrapie and scrapie-negative sheep. It was found that PrPSc accumulated in the enteric nervous system (ENS) of all scrapie-infected sheep but not in scrapie-negative sheep. The distribution of PrPSc within the ENS was then studied along the entire gastro-intestinal tract in seven scrapie-infected sheep carrying various PrP genotypes. In sheep with the highest genetically determined susceptibility to scrapie, PrPSc was detected in the ENS from the oesophagus to the rectum. In sheep with a lower genetic susceptibility to scrapie, PrPSc was present in the ENS of the forestomachs, small intestine and large intestine but not in the oesophagus. In a scrapie-negative sheep with a PrP genotype associated with scrapie resistance, no PrPSc was seen in the ENS at any site along the gastro-intestinal tract. The presence of PrPSc within the ENS of scrapie-infected sheep indicates a possible role of the ENS in the pathogenesis of natural scrapie as a portal of entry to the central nervous system.

L10 ANSWER 13 OF 21 CABA COPYRIGHT 2004 CABI on STN

AN 1998:112387 CABA
DN 19982210900
TI Tonsillar biopsy and PrPSc detection in the preclinical diagnosis of scrapie
AU Schreuder, B. E. C.; Keulen, L. J. M. van; Vromans, M. E. W.; Langeveld, J. P. M.; Smits, M. A.; ***Van Keulen, L. J. M.***
CS Institute for Animal Science and Health (ID-DLO) PO Box 65, 8200 AB Lelystad, Netherlands.
SO Veterinary Record, (1998) Vol. 142, No. 21, pp. 564-568. 31 ref.
ISSN: 0042-4900
DT Journal
LA English
ED Entered STN: 19980714
Last Updated on STN: 19980714
AB Preliminary findings have indicated that in naturally infected sheep, fully susceptible to scrapie (VRQ-homozygous), PrPSc can be detected in the tonsils approximately one year before the expected onset of clinical disease, whereas no immunostaining can be detected in animals with a semi-resistant genotype. This paper describes the technique for taking tonsillar biopsies from sheep. In another experiment PrPSc was detected even earlier in comparable VRQ-homozygous sheep born and raised in different surroundings. At three-and-a-half months of age no PrPSc could be detected in 3 homozygous susceptible sheep (VRQ/VRQ), but PrPSc was detected at 4 months in one similar sheep. At 8 months of age all 7 sampled VRQ/VRQ sheep showed positive immunostaining in the biopsies, but none of the biopsies from three VRQ/ARQ heterozygotes showed any immunostaining; they were positive when sampled at 14 to 15 months of age. Biopsies from VRQ/ARR sheep were negative throughout this period. On the basis of the established or expected incubation period, PrPSc could thus be detected in the tonsils of live susceptible animals at between one-third and a half of the incubation period, more than one-and-a-half years before clinical signs normally appear in both these genotypes.

L10 ANSWER 14 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1997:679268 CAPLUS
DN 127:316560
TI Method for the detection of ***prion*** diseases
IN Schreuder, Bram Edward Cornelis; ***Van Keulen, Lucius Johannes*** *** Mattheus*** ; Vromans, Maria Elisabeth Wilhelmina; Langeveld, Johannes Pieter Maria; Smits, Marinus Adrianus
PA Instituut voor Dierhouderij en Diergezondheid (Id-Dlo), Neth.; Schreuder, Bram Edward Cornelis; Van Keulen, Lucius Johannes Mattheus; Vromans, Maria Elisabeth Wilhelmina; Langeveld, Johannes Pieter Maria; Smits, Marinus Adrianus
SO PCT Int. Appl., 29 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9737227	A1	19971009	WO 1997-NL166	19970402
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2250800	AA	19971009	CA 1997-2250800	19970402
AU 9721808	A1	19971022	AU 1997-21808	19970402
AU 713529	B2	19991202		
EP 891552	A1	19990120	EP 1997-914658	19970402
EP 891552	B1	20030402		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
BR 9708421	A	19990803	BR 1997-8421	19970402
NZ 332132	A	20000228	NZ 1997-332132	19970402
JP 2000505559	T2	20000509	JP 1997-535157	19970402
JP 3333213	B2	20021015		

	AT 236407	E	20030415	AT 1997-914658	19970402
	NO 9804602	A	19981203	NO 1998-4602	19981001
PRAI	EP 1996-200917	A	19960403		
	WO 1997-NL166	W	19970402		

AB The invention provides methods for the detection of ***prion*** diseases, such as scrapie of sheep, bovine spongiform encephalopathy of cattle, Creutzfeld-Jacob disease of man, whereby aberrant proteins or ***prion*** proteins are detected in tissues which can be sampled from live animals. Peptides such as segments of the scrapie protein can be used to raise antibodies for use in immunoassays of lymphoid tissues such as the tonsils.

L10 ANSWER 15 OF 21 CABA COPYRIGHT 2004 CABI on STN
AN 97:153958 CABA
DN 19972216710
TI Control of scrapie eventually possible?
AU Schreuder, B. E. C.; Keulen, L. J. M. van; Smits, M. A.; Langeveld, J. P. M.; Stegeman, J. A.; ***Van Keulen, L. J. M.***
CS DLO-Institute for Animal Science and Health (IDO-DLO), Research Head Office, P.O. Box 65, 8200 AB Lelystad, Netherlands.
SO Veterinary Quarterly, (1997) Vol. 19, No. 3, pp. 105-113. 43 ref.
ISSN: 0165-2176
DT Journal
LA English
ED Entered STN: 19971211
Last Updated on STN: 19971211

L10 ANSWER 16 OF 21 CABA COPYRIGHT 2004 CABI on STN
AN 96:130399 CABA
DN 19962212842
TI Preclinical test for ***prion*** diseases
AU Schreuder, B. E. C.; Keulen, L. J. M. van; Vromans, M. E. W.; Langeveld, J. P. M.; Smits, M. A.; ***Van Keulen, L. J. M.***
CS DLO-Institute for Animal Science and Health (ID-DLO), PO Box 65, 8200 AB Lelystad, Netherlands.
SO Nature (London), (1996) Vol. 381, No. 6583, pp. 563. 10 ref.
ISSN: 0028-0836
DT Letter
LA English
ED Entered STN: 19961015
Last Updated on STN: 19961015
AB Tonsillar samples were taken from ten 9.5- to 10-month-old lambs, born and maintained on a farm infected with scrapie. None of the sheep showed clinical signs of the disease. However, extensive PrPSc (an altered protein associated with ***prion*** encephalopathies) immunostaining was found in these biopsies from 6 susceptible sheep with the genotype PrPVQ/VQ. PrPSc was not detected in the other sheep which were of the resistant genotype PrPVQ/AR. It is concluded that screening tonsillar tissue for PrPSc by immunohistochemistry offers a potential method of preclinical diagnosis of scrapie in sheep.

L10 ANSWER 17 OF 21 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 8
AN 1996:270052 BIOSIS
DN PREV199698826181
TI Immunohistochemical detection of ***prion*** protein in lymphoid tissues of sheep with natural scrapie.
AU ***Van Keulen, L. J. M.*** [Reprint author]; Schreuder, B. E. C.; Meloen, R. H.; Mooij-Harkes, G.; Vromans, M. E. W.; Langeveld, J. P. M.
CS Dep. Pathobiol. Epidemiol., Inst. Anim. Sci. Health, PO Box 65, 8200 AB Lelystad, Netherlands
SO Journal of Clinical Microbiology, (1996) Vol. 34, No. 5, pp. 1228-1231.
CODEN: JCMIDW. ISSN: 0095-1137.
DT Article
LA English
ED Entered STN: 10 Jun 1996
Last Updated on STN: 10 Jun 1996
AB The scrapie-associated form of the ***prion*** protein (PrP-Sc) accumulates in the brain and lymphoid tissues of sheep with scrapie. In order to assess whether detecting PrP-Sc in lymphoid tissue could be used as a diagnostic test for scrapie, we studied the localization and

distribution of PrP-Sc in various lymphoid tissues collected at necropsy from 55 sheep with clinical scrapie. Samples collected from the spleen, palatine tonsil, ileum, and five different lymph nodes were immunohistochemically stained for PrP-Sc cntdot PrP-Sc was found to be deposited in a reticular pattern in the center of both primary and secondary lymphoid follicles. In addition, granules of PrP-Sc were seen in the cytoplasm in macrophages associated with the lymphoid follicles. In 54 (98%) of the 55 scrapie-affected sheep, PrP-Sc was detected in the spleen, retropharyngeal lymph node, mesenteric lymph node, and the palatine tonsil. However, only in the palatine tonsils was PrP-Sc present in a consistently high percentage of the lymphoid follicles. PrP was not detected in any of the lymphoid tissues of 12 sheep that had no neurohistopathological signs of a scrapie infection. We conclude that the tonsils are the best-suited lymphoid tissue to be biopsied for the detection of PrP-Dc in the diagnosis of clinical scrapie in living sheep.

L10 ANSWER 18 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 9
AN 1996:358658 CAPLUS
TI Preclinical test for ***prion*** diseases
AU Schreuder, B. E. C.; ***van Keulen, L. J. M.*** ; Vromans, M. E. W.; Langeveld, J. P. M.; Smits, M. A.
CS DLO-Inst. Anim. Sci. Health, Leylstad, 8200 AB, Neth.
SO Nature (London) (1996), 381(6583), 563
CODEN: NATUAS; ISSN: 0028-0836
PB Macmillan Magazines
DT Journal; Letter
LA English
AB Unavailable

L10 ANSWER 19 OF 21 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 96182917 EMBASE
DN 1996182917
TI Preclinical test for ***prion*** diseases [2].
AU Schreuder B.E.C.; ***Van Keulen L.J.M.*** ; Vromans M.E.W.; Langeveld J.P.M.; Smits M.A.
CS DLO-Inst Animal Sci Health (ID-DLO), PO Box 65,8200 AB Lelystad, Netherlands
SO Nature, (1996) 381/6583 (563).
ISSN: 0028-0836 CODEN: NATUAS
CY United Kingdom
DT Journal; Letter
FS 004 Microbiology
008 Neurology and Neurosurgery
LA English

L10 ANSWER 20 OF 21 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 96328470 EMBASE
DN 1996328470
TI [***Prion*** diseases of humans and animals].
PRIONZIEKTEN BIJ MENSEN EN DIEREN.
AU Smits M.A.; Schreuder B.E.C.; ***Van Keulen L.J.M.*** ; Langeveld J.P.M.
CS ID-DLO, Postbus 65,8200 AB Lelystad, Netherlands
SO Nederlands Tijdschrift voor Medische Microbiologie, (1996) 4/2 (30-34).
ISSN: 0929-0176 CODEN: NMMIEB
CY Netherlands
DT Journal; Article
FS 004 Microbiology
LA Dutch
SL Dutch; English
AB In the recent years important progress has been made in the research on ***prion*** diseases. Nevertheless there are important gaps in our knowledge on ***prion*** diseases. The nature of the infectious agent is unknown, insight into the risk factors is limited, the possibilities to diagnose ***prion*** diseases are not adequate, and there are no therapeutic approaches available. The lack of sufficient scientific knowledge has contributed to the mad cow disease crisis in March this year.

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L10  ANSWER 21 OF 21  CABA COPYRIGHT 2004 CABI on STN
AN   95:132740  CABA
DN   19952210471
TI   Immunohistochemical detection and localization of   ***prion***   protein
     in brain tissue of sheep with natural scrapie
AU   Keulen, L. J. M. van; Schreuder, B. E. C.; Meloen, R. H.; Berg, M. P. van
     den; Mooij-Harkes, G.; Vromans, M. E. W.; Langeveld, J. P. M.;   ***Van***
  ***     Keulen, L. J. M.***  ; Van den Berg, M. P.
CS   Department of Pathobiology, Central Veterinary Institute, Institute for
     Animal Science and Health (ID-DLO), 8200 AB Lelystad, Netherlands.
SO   Veterinary Pathology, (1995) Vol. 32, No. 3, pp. 299-308. 35 ref.
     ISSN: 0300-9858
DT   Journal
LA   English
ED   Entered STN: 19950724
     Last Updated on STN: 19950724
AB   Tissue samples from the brains of 50 sheep with natural scrapie and 20
     sheep without histopathological signs of scrapie were treated with formic
     acid and hydrated autoclaving. A scrapie-associated cellular   ***prion***
     protein (PrPSC) was detected using an antipeptide antisera. PrPSC was
     located in the brains of all sheep with scrapie; no immunostaining
     occurred in sheep without scrapie. PrPSC that did not stain for amyloid
     was present in the cytoplasm and at the cell membrane of neurons and
     astrocytes. Large amounts of PrPSC were seen at the cell membrane of
     neurons in the medulla oblongata and pons, whereas PrPSC accumulated at
     the cell membrane of astrocytes of the glial limitans in all brain
     regions. PrPSC that stained for amyloid was located in the walls of blood
     vessels and perivascularly in the brains of 64% of the sheep. No apparent
     topographic relationship existed between PrPSC that stained for amyloid
     and PrPSC accumulation associated with neurons or astrocytes. In all
     scrapie-affected sheep PrPSC was present in brain regions with
     vacuolation, but it could also be detected in regions with little or no
     vacuolation.


=> e schreuder bram/au
E1            3     SCHREUDER BAS J N/AU
E2            1     SCHREUDER BERT/AU
E3            3 --> SCHREUDER BRAM/AU
E4            6     SCHREUDER BRAM E C/AU
E5            2     SCHREUDER BRAM EDWARD CORNELIS/AU
E6           20     SCHREUDER C/AU
E7           22     SCHREUDER C H/AU
E8            2     SCHREUDER COR/AU
E9            2     SCHREUDER D/AU
E10           4     SCHREUDER D A/AU
E11           1     SCHREUDER D R/AU
E12           1     SCHREUDER DIRK J/AU

=> s e3-e5
L11          11 ("SCHREUDER BRAM"/AU OR "SCHREUDER BRAM E C"/AU OR "SCHREUDER
                BRAM EDWARD CORNELIS"/AU)

=> dup rem l11
PROCESSING COMPLETED FOR L11
L12           8 DUP REM L11 (3 DUPLICATES REMOVED)

=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 8 ANSWERS - CONTINUE? Y/(N):y

L12  ANSWER 1 OF 8  BIOSIS  COPYRIGHT (c) 2004 The Thomson Corporation.  on STN
     DUPLICATE 1
AN   2004:143248  BIOSIS
DN   PREV200400143547
TI   Quantifying BSE control by calculating the basic reproduction ratio R0 for
     the infection among cattle.
AU   de Koeijer, Aline [Reprint Author]; Heesterbeek, Hans [Reprint Author];
       ***Schreuder, Bram***   [Reprint Author]; Oberthuer, Radulf; Wilesmith,
     John; van Roermund, Herman [Reprint Author]; de Jong, Mart [Reprint
     Author]
CS   Institute for Animal Science and Health, ID-Lelystad, 6200 AB, P.O. Box
```

65, Lelystad, Netherlands
a.a.dekoeijer@id.dlo.nl
SO Journal of Mathematical Biology, (January 2004) Vol. 48, No. 1, pp. 1-22. print.
ISSN: 0303-6812.
DT Article
LA English
ED Entered STN: 10 Mar 2004
Last Updated on STN: 10 Mar 2004
AB The safety of using meat and bone meal (MBM) in mammal feed was studied in view of BSE, by quantifying the risk of BSE transmission through different infection routes. This risk is embodied in the basic reproduction ratio R0 of the infection, i.e. the average number of new infections induced by one initial infection. Only when R0 is below 1, will the disease die out with certainty and the population will become free from BSE. Unfortunately this is a slow process due to the slow progression of the disease. We calculate R0 explicitly from basic ingredients taking several different transmission routes into account. Several of the basic ingredients are functions of age or of infection-age. We also calculate the exponential growth rate r in terms of the same basic ingredients. Next we quantify the ingredients from available data and compute the effects on R0 of various scenario's for controlling BSE, with examples for the UK and the Netherlands.

L12 ANSWER 2 OF 8 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 2003:100080 BIOSIS
DN PREV200300100080
TI Factors that influence the age distribution of BSE cases: Potentials for age targeting in surveillance.
AU De Koeijer, Aline [Reprint Author]; ***Schreuder, Bram*** ; Bouma, Annemarie
CS Division of Infectious Diseases and Food Chain Quality, Institute for Animal Science and Health (ID-Lelystad), 8200 AB, P.O. Box 65, Lelystad, Netherlands
a.a.dekoeijer@id.wag-ur.nl
SO Livestock Production Science, (September 2002) Vol. 76, No. 3, pp. 223-233. print.
ISSN: 0301-6226 (ISSN print).
DT Article
LA English
ED Entered STN: 19 Feb 2003
Last Updated on STN: 19 Feb 2003
AB Recently, due to consumers fears concerning BSE and vCJD, the need arose for methods to detect BSE, to estimate the present prevalence of BSE among cattle and to predict future BSE prevalence. As a part of that set of urgent questions, it has become important to indicate groups in which BSE risk is higher or lower. One of the well-known risk factors for BSE is age: very young animals do not develop the disease, and very old animals are less likely to develop the disease. Using age-structured modelling, three factors influencing the age distribution of BSE were found to be important: (1) the incubation period of BSE, (2) age structure of the cattle population, and (3) the local risk history (methods of rendering, feeding of compound feed containing Meat and Bone Meal (MBM), and the development of BSE control). The EU has considered these three risk factors to be the most important for BSE risk assessment. So far, this EU risk assessment method has been proven right by several countries detecting BSE after being classified as 'BSE is most likely present here'. The age distribution of BSE seems to vary a lot between countries and regions. When information on these three factors is available, the expected age distribution of BSE in different countries can be calculated. Our calculations show that in countries where, until very recently, the reproduction ratio was high, (i.e., BSE risk factors were high), the BSE prevalence is expected to be highest in 4-year-old cattle. In countries with low reproduction ratio for BSE, (i.e., BSE control at a very high level) for more than 5 years, the prevalence will be highest in the 6-8-year-old cattle. Thus, surveillance could be targeted specifically at the age groups with the highest BSE risk. For each country, a short assessment shows in which age group BSE is most likely to be found.

L12 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:574024 CAPLUS

DN 133:174276
TI Prion test using guanidine thiocyanate for reducing false positive test results
IN Garssen, Gerrit Jan; Jacobs, Jorg Gunther; Langeveld, Joannes Pieter Maria; Smits, Marinus Adrianus; Van Keulen, Lucien Johannes Mattheus; ***Schreuder, Bram Edward Cornelis*** ; Bossers, Alexander
PA Stichting Dienst Landbouwkundig Onderzoek, Neth.
SO PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000048003	A1	20000817	WO 2000-NL79	20000209
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1151305	A1	20011107	EP 2000-904139	20000209
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
PRAI EP 1999-200391	A	19990211		
WO 2000-NL79	W	20000209		

AB The invention is related to diagnostic methods for detecting transmissible spongiform encephalopathies (TSEs) such as BSE and scrapie and related disease in humans. The invention provides use of guanidine thiocyanate (gdnSCN) or a functional equiv. thereof for treating at least one sample derived from a mammal, including humans for reducing the risk of scoring a false-pos. test result in testing said sample for the presence or absence of aberrant prion protein.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1997:679268 CAPLUS
DN 127:316560
TI Method for the detection of prion diseases
IN ***Schreuder, Bram Edward Cornelis*** ; Van Keulen, Lucius Johannes Mattheus; Vromans, Maria Elisabeth Wilhelmina; Langeveld, Johannes Pieter Maria; Smits, Marinus Adrianus
PA Instituut voor Dierhouderij en Diergezondheid (Id-Dlo), Neth.; Schreuder, Bram Edward Cornelis; Van Keulen, Lucius Johannes Mattheus; Vromans, Maria Elisabeth Wilhelmina; Langeveld, Johannes Pieter Maria; Smits, Marinus Adrianus
SO PCT Int. Appl., 29 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9737227	A1	19971009	WO 1997-NL166	19970402
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
CA 2250800	AA	19971009	CA 1997-2250800	19970402
AU 9721808	A1	19971022	AU 1997-21808	19970402
AU 713529	B2	19991202		
EP 891552	A1	19990120	EP 1997-914658	19970402
EP 891552	B1	20030402		

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

	BR 9708421	A	19990803	BR 1997-8421	19970402
	NZ 332132	A	20000228	NZ 1997-332132	19970402
	JP 2000505559	T2	20000509	JP 1997-535157	19970402
	JP 3333213	B2	20021015		
	AT 236407	E	20030415	AT 1997-914658	19970402
	NO 9804602	A	19981203	NO 1998-4602	19981001
PRAI	EP 1996-200917	A	19960403		
	WO 1997-NL166	W	19970402		

AB The invention provides methods for the detection of prion diseases, such as scrapie of sheep, bovine spongiform encephalopathy of cattle, Creutzfeld-Jacob disease of man, whereby aberrant proteins or prion proteins are detected in tissues which can be sampled from live animals. Peptides such as segments of the scrapie protein can be used to raise antibodies for use in immunoassays of lymphoid tissues such as the tonsils.

L12 ANSWER 5 OF 8 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 2
AN 1996:526174 BIOSIS
DN PREV199699248530
TI PrP genotype contributes to determining survival times of sheep with natural scrapie.
AU Bossers, Alex; ***Schreuder, Bram E. C.*** ; Muileman, Ida H.; Belt, Peter B. G. M.; Smits, Mari A. [Reprint author]
CS Dep. Bacteriol., DLO-Inst. Anim. Sci. Health, P.O. Box 65, 8200 AB Lelystad, Netherlands
SO Journal of General Virology, (1996) Vol. 77, No. 10, pp. 2669-2673. CODEN: JGVIAY. ISSN: 0022-1317.
DT Article
LA English
ED Entered STN: 22 Nov 1996
Last Updated on STN: 22 Nov 1996
AB Several allelic variants of the sheep PrP gene are associated with scrapie susceptibility. However, it is not known whether, and to what extent, the PrP genotype contributes to determining survival times of scrapie sheep. We therefore determined the PrP genotype and life spans of over 50 Flemish and Swifter sheep within a single scrapie-affected flock. Eighty-three per cent of the scrapie sheep were homozygous for the PrPvQ allele (polymorphic amino acids at codons 136 and 171 are indicated) and these sheep died from scrapie at a mean age of 25 months. In sheep heterozygous for PrP-VQ, development of scrapie was delayed or did not occur. Sheep with at least one PrP-AR allele, including PrP-VQ/PrP-AR sheep, did not develop scrapie. No scrapie sheep were found without a PrP-VQ allele. We conclude that the PrP genotype contributes to determining survival times of sheep with natural scrapie. Additionally, we describe two novel sheep PrP allelic variants.

L12 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1997:593179 CAPLUS
DN 127:246402
TI PrP allelic variants associated with natural scrapie
AU Belt, Peter B. G. M.; Bossers, Alex; ***Schreuder, Bram E. C.*** ; Smits, Mari A.
CS Department of Bacteriology, Institute for Animal Science and Health (ID-DLO), Lelystad, Neth.
SO Bovine Spongiform Encephalopathy: The BSE Dilemma, [Proceedings of the International Workshop on Bovine Spongiform Encephalopathy: The BSE Dilemma], 6th, Williamsburg, Va., Feb. 26-Mar. 1, 1995 (1996), 294-305. Editor(s): Gibbs, Clarence J. Publisher: Springer, New York, N. Y. CODEN: 64ZIAF
DT Conference
LA English
AB The PrP allelic variants of 69 scrapie-affected and 176 healthy sheep by denaturing gradient gel electrophoresis (DGGE) were detd. The results indicated that specific combinations of polymorphisms within the PrP gene of sheep, rather that single polymorphisms, are assocd. with the incidence of scrapie. A pos. selection for the PrPARR allele and/or a neg. selection for the PrPVRQ in breeding programs could help to control natural scrapie.

L12 ANSWER 7 OF 8 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 3
AN 1995:205822 BIOSIS
DN PREV199598220122
TI Identification of five allelic variants of the sheep PrP gene and their association with natural scrapie.
AU Belt, Peter B. G. M. [Reprint author]; Muileman, Ida H.; ***Schreuder,*** *** Bram E. C.*** ; Ruijter, Judy Bos-De; Gielkens, Arno L. J.; Smits, Mari A.
CS Dep. Mol. Biol., DLO-Inst. Anim. Sci. Health, Edelhertweg 15, 8219 PH Lelystad, Netherlands
SO Journal of General Virology, (1995) Vol. 76, No. 3, pp. 509-517.
CODEN: JGVIAY. ISSN: 0022-1317.
DT Article
LA English
ED Entered STN: 23 May 1995
Last Updated on STN: 23 May 1995
AB Scrapie is a fatal neurodegenerative disease of sheep that belongs to the group of prion diseases found in humans and animals. The host encoded prion protein (PrP) plays a central role in the disease process. In the PrP genes of man, mice and sheep, polymorphisms have been found that are associated with disease susceptibility and pathogenesis. We have used denaturing gradient gel electrophoresis (DGGE) to detect polymorphisms in the sheep PrP gene. In addition to the already described polymorphisms at codons 136, 154 and 171, we identified a hitherto unknown G fwdarw T transition at codon 171. This transition is responsible for a glutamine to histidine substitution. An arginine to glutamine substitution at this position has been described previously. DGGE allowed us to identify five different combinations of these polymorphisms within the PrP gene representing five allelic variants, which were cloned and sequenced. Based on the triplet sequences present at codons 136, 154 and 171 these allelic variants were designated PrP-VRQ, PrP-ARR, PrP-ARQ, PrP-ARH and PrP-AHQ. To determine the association of these allelic variants with natural scrapie, we screened 34 scrapie affected and 91 healthy control sheep of the Texel breed for the presence of these allelic variants. In these two groups, the five variants gave rise to 13 different genotypes. The distribution of the allelic variants among both groups showed marked differences. The PrP-VRQ variant was present with high frequency in scrapie affected sheep, whereas the PrP-ARR variant was almost exclusively present in the healthy group. Two other variants, PrP-ARQ and PrP-ARH, were found in both groups with equal frequencies. The data obtained suggest modulation of disease susceptibility in these Texel sheep by at least five different PrP allelic variants, with the PrP-VRQ and PrP-ARR alleles acting in a dominant, but opposite fashion over the PrP-ARQ and PrP-ARH alleles. The frequency of the PrP-AHQ variant was too low to draw any conclusions.

L12 ANSWER 8 OF 8 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1994:212451 BIOSIS
DN PREV199497225451
TI BSE agent hypotheses.
AU ***Schreuder, Bram E. C.***
CS DVM, DLO-Central Vet. Inst., P.O. Box 65, 8200 AB Lelystad, Netherlands
SO Livestock Production Science, (1994) Vol. 38, No. 1, pp. 23-33.
CODEN: LPSCDL. ISSN: 0301-6226.
DT Article
LA English
ED Entered STN: 10 May 1994
Last Updated on STN: 10 May 1994
AB Although the oldest known form of transmissible spongiform encephalopathy (TSE), scrapie in sheep, has been described as early as in 1732, the nature of the agents causing TSEs still remains an enigma. Unusual properties of the agents, such as extreme resistance against UV, ionizing radiation, and dry heat, already led to the term "unconventional virus". From the number of hypotheses on the nature of these agents postulated, we will consider here three major ones only: virus, virino and prion. Present schools of thought, however, confine themselves mainly to the last two hypotheses. A recently formulated unified theory tries to reconcile the essentials of these two hypotheses. The virino hypothesis was called upon essentially to explain the variability in scrapie isolates when

passaged in experimental rodents. Nucleic acids in a micro-organism would form the obvious explanation for such variability, but they have not been identified to date, even by using promising modem recombinant DNA methodologies. Research supporting the prion hypothesis has progressed steadily since its formulation. There was the discovery of the so-called prion protein (PrP), encoded by a single host gene. This PrP-gene is transcribed both in scrapie-infected animals and in normal animals. The resulting prion protein in its normal form was designated PrP-C, meaning cellular PrP. The abnormal form, found in infected animals only, was designated PrP-Sc, meaning scrapie PrP. The isoforms are not identical, though they seem to have the same primary structure. Supporters of both the virino and the prion hypothesis equally accept that PrP is a key element in the pathogenesis of these diseases. Proposed models, involving the conversion of the normal PrP-C into PrP-Sc as part of the pathogenesis, will be discussed. Further experimental support for the prion theory has recently been obtained through work with transgenic animals.

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=> e bossers alexander/au
E1            7     BOSSERS A M/AU
E2           15     BOSSERS ALEX/AU
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E4            1     BOSSERS ANN/AU
E5           21     BOSSERS B/AU
E6            6     BOSSERS BERNADETTE/AU
E7            4     BOSSERS CHRIS F/AU
E8            5     BOSSERS G T/AU
E9           10     BOSSERS G T M/AU
E10           1     BOSSERS J M/AU
E11           2     BOSSERS P A/AU
E12           1     BOSSERS PIETER A/AU

=> s e1-e3 and prion?
L13          16 ("BOSSERS A M"/AU OR "BOSSERS ALEX"/AU OR "BOSSERS ALEXANDER"/AU
                ) AND PRION?

=> dup rem l13
PROCESSING COMPLETED FOR L13
L14           9 DUP REM L13 (7 DUPLICATES REMOVED)

=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 9 ANSWERS - CONTINUE? Y/(N):y

L14  ANSWER 1 OF 9  BIOSIS  COPYRIGHT (c) 2004 The Thomson Corporation.  on STN
     DUPLICATE 1
AN   2004:77483  BIOSIS
DN   PREV200400079379
TI   Enzymatic degradation of   ***prion***   protein in brain stem from
     infected cattle and sheep.
AU   Langeveld, Jan P. M. [Reprint Author]; Wang, Jeng-Jie; van de Wiel, Dick
     F. M.; Shih, Giles C.; Garssen, G. Jan;   ***Bossers, Alex***  ; Shih,
     Jason C. H.
CS   Central Institute for Animal Disease Control, 8203 AA, PO Box 2004,
     Lelystad, Netherlands
     jan.langeveld@wur.nl
SO   Journal of Infectious Diseases, (1 December 2003) Vol. 188, No. 11, pp.
     1782-1789. print.
     CODEN: JIDIAQ. ISSN: 0022-1899.
DT   Article
LA   English
ED   Entered STN: 4 Feb 2004
     Last Updated on STN: 4 Feb 2004
AB     ***Prions***  -infectious agents involved in transmissible spongiform
     encephalopathies-normally survive proteolytic and mild protein-destructive
     processes. Using bacterial keratinase produced by Bacillus licheniformis
     strain PWD-1, we tested conditions to accomplish the full degradation of
       ***prion***   protein (PrP) in brain-stem tissue from animals with bovine
     spongiform encephalopathy and scrapie. The detection of PrPSc, the
     disease-associated isoform of PrP, in homogenates was done by Western
     blotting and various antibodies. The results indicated that only in the
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presence of detergents did heat pretreatment at >100degreeC allow the extensive enzymatic breakdown of PrPSc to a state where it is immunochemically undetectable. Proteinase K and 2 other subtilisin proteases, but not trypsin and pepsin, were also effective. This enzymatic process could lead to the development of a method for the decontamination of medical and laboratory equipment. The ultimate effectiveness of this method of ***prion*** inactivation has to be tested in mouse bioassays.

L14 ANSWER 2 OF 9 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 2
AN 2003:248861 BIOSIS
DN PREV200300248861
TI In vitro conversion of normal ***prion*** protein into pathologic isoforms.
AU ***Bossers, Alex*** [Reprint Author]; Rigter, Alan; de Vries, Ruth; Smits, Mari A.
CS Central Institute for Animal Disease Control (CIDC-Lelystad), Edelhertweg 15, 8219 PH, Lelystad, Netherlands
a.bossers@id.dlo.nl
SO Clinics in Laboratory Medicine, (March 2003) Vol. 23, No. 1, pp. 227-247. print.
ISSN: 0272-2712.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 21 May 2003
Last Updated on STN: 21 May 2003

L14 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:574024 CAPLUS
DN 133:174276
TI ***Prion*** test using guanidine thiocyanate for reducing false positive test results
IN Garssen, Gerrit Jan; Jacobs, Jorg Gunther; Langeveld, Joannes Pieter Maria; Smits, Marinus Adrianus; Van Keulen, Lucien Johannes Mattheus; Schreuder, Bram Edward Cornelis; ***Bossers, Alexander***
PA Stichting Dienst Landbouwkundig Onderzoek, Neth.
SO PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000048003	A1	20000817	WO 2000-NL79	20000209
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1151305	A1	20011107	EP 2000-904139	20000209
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
PRAI EP 1999-200391	A	19990211		
WO 2000-NL79	W	20000209		

AB The invention is related to diagnostic methods for detecting transmissible spongiform encephalopathies (TSEs) such as BSE and scrapie and related disease in humans. The invention provides use of guanidine thiocyanate (gdnSCN) or a functional equiv. thereof for treating at least one sample derived from a mammal, including humans for reducing the risk of scoring a false-pos. test result in testing said sample for the presence or absence of aberrant ***prion*** protein.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 4 OF 9 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

DUPLICATE 3
AN 2000:106273 BIOSIS
DN PREV200000106273
TI Susceptibility of sheep for scrapie as assessed by in vitro conversion of nine naturally occurring variants of PrP.
AU ***Bossers, Alex*** ; de Vries, Ruth; Smits, Mari A. [Reprint author]
CS Institute for Animal Science and Health, Lelystad, Netherlands
SO Journal of Virology, (Feb., 2000) Vol. 74, No. 3, pp. 1407-1414. print.
CODEN: JOVIAM. ISSN: 0022-538X.
DT Article
LA English
ED Entered STN: 22 Mar 2000
Last Updated on STN: 3 Jan 2002
AB Polymorphisms in the ***prion*** protein (PrP) gene are associated with phenotypic expression differences of transmissible spongiform encephalopathies in animals and humans. In sheep, at least 10 different mutually exclusive polymorphisms are present in PrP. In this study, we determined the efficiency of the in vitro formation of protease-resistant PrP of nine sheep PrP allelic variants in order to gauge the relative susceptibility of sheep for scrapie. No detectable spontaneous protease-resistant PrP formation occurred under the cell-free conditions used. All nine host-encoded cellular PrP (PrPC) variants had distinct conversion efficiencies induced by PrPSc isolated from sheep with three different homozygous PrP genotypes. In general, PrP allelic variants with polymorphisms at either codon 136 (Ala to Val) or codon 141 (Leu to Phe) and phylogenetic wild-type sheep PrPC converted with highest efficiency to protease-resistant forms, which indicates a linkage with a high susceptibility of sheep for scrapie. PrPC variants with polymorphisms at codons 171 (Gln to Arg), 154 (Arg to His), and to a minor extent 112 (Met to Thr) converted with low efficiency to protease-resistant isoforms. This finding indicates a linkage of these alleles with a reduced susceptibility or resistance for scrapie. In addition, PrPSc with the codon 171 (Gln-to-His) polymorphism is the first variant reported to induce higher conversion efficiencies with heterologous rather than homologous PrP variants. The results of this study strengthen our views on polymorphism barriers and have further implications for scrapie control programs by breeding strategies.

L14 ANSWER 5 OF 9 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 4
AN 1997:260741 BIOSIS
DN PREV199799567344
TI Scrapie susceptibility-linked polymorphisms modulate the in vitro conversion of sheep ***prion*** protein to protease-resistant forms.
AU ***Bossers, Alex*** ; Belt, Peter B. G. M.; Raymond, Gregory J.; Caughey, Byron; De Vries, Ruth; Smits, Mari A. [Reprint author]
CS Dep. Bacteriol., DLO-Inst. Animal Sci. Health, P.O. Box b5, 8200 AB Lelystad, Netherlands
SO Proceedings of the National Academy of Sciences of the United States of America, (1997) Vol. 94, No. 10, pp. 4931-4936.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
LA English
ED Entered STN: 24 Jun 1997
Last Updated on STN: 24 Jun 1997
AB ***Prion*** diseases are natural transmissible neurodegenerative disorders in humans and animals. They are characterized by the accumulation of a protease-resistant scrapie-associated ***prion*** protein (PrP-Sc) of the host-encoded cellular ***prion*** protein (PrP-C) mainly in the central nervous system. Polymorphisms in the PrP gene are linked to differences in susceptibility for ***prion*** diseases. The mechanisms underlying these effects are still unknown. Here we describe studies of the influence of sheep PrP polymorphisms on the conversion of PrP-C into protease-resistant forms. In a cell-free system, sheep PrP-Sc induced the conversion of sheep PrP-C into protease-resistant PrP (PrP-res) similar or identical to PrP-Sc. Polymorphisms present in either PrP-C or PrP-Sc had dramatic effects on the cell-free conversion efficiencies. The PrP variant associated with a high susceptibility to scrapie and short survival times of scrapie-affected sheep was efficiently converted into PrP-res. The wild-type PrP variant associated with a neutral effect on susceptibility

and intermediate survival times was converted with intermediate efficiency. The PrP variant associated with scrapie resistance and long survival times was poorly converted. Thus the in vitro conversion characteristics of the sheep PrP variants reflect their linkage with scrapie susceptibility and survival times of scrapie-affected sheep. The modulating effect of the polymorphisms in PrPc and PrPsc on the cell-free conversion characteristics suggests that, besides the species barrier, polymorphism barriers play a significant role in the transmissibility of ***prion*** diseases.

L14 ANSWER 6 OF 9 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 1997:422684 BIOSIS
DN PREV199799721887
TI Molecular assessment of human susceptibility to BSE and scrapie.
AU Raymond, Gregory [Reprint author]; Hope, James; Kocisko, David [Reprint author]; Priola, Suzette [Reprint author]; Raymond, Lynne [Reprint author]; ***Bossers, Alex*** ; Lansbury, Peter; Caughey, Byron [Reprint author]
CS NIH/NIAID, Rocky Mountain Lab., Rocky Mountain, MN, USA
SO FASEB Journal, (1997) Vol. 11, No. 9, pp. A1441.
Meeting Info.: 17th International Congress of Biochemistry and Molecular Biology in conjunction with the Annual Meeting of the American Society for Biochemistry and Molecular Biology. San Francisco, California, USA. August 24-29, 1997.
CODEN: FAJOEC. ISSN: 0892-6638.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 8 Oct 1997
Last Updated on STN: 8 Oct 1997

L14 ANSWER 7 OF 9 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 5
AN 1997:353905 BIOSIS
DN PREV199799660308
TI Molecular assessment of the potential transmissibilities of BSE and scrapie to humans.
AU Raymond, Gregory J. [Reprint author]; Hope, James; Kocisko, David A. [Reprint author]; Priola, Suzette A. [Reprint author]; Raymond, Lynne D. [Reprint author]; ***Bossers, Alex*** ; Ironside, James; Will, Robert G.; Chen, Shu G.; Petersen, Robert B.; Gambetti, Pierluigi; Rubenstein, Richard; Smits, Mari A.; Lansbury., Peter T., Jr.; Caughey, Bryon [Reprint author]
CS Rocky Mountain Lab., NIAID, National Inst. Health, Hamilton, MT 59840, USA
SO Nature (London), (1997) Vol. 388, No. 6639, pp. 285-288.
CODEN: NATUAS. ISSN: 0028-0836.
DT Article
LA English
ED Entered STN: 25 Aug 1997
Last Updated on STN: 25 Aug 1997
AB More than a million cattle infected with bovine spongiform encephalopathy (BSE) may have entered the human food chain'. Fears that BSE might transmit to man were raised when atypical cases of Creutzfeldt-Jakob disease (CJD), a human transmissible spongiform encephalopathy (TSE), emerged in the UK. In BSE and other TSE diseases, the conversion of the protease-sensitive host ***prion*** protein (PrP-sen) to a protease-resistant isoform (PrP-res) is an important event in pathogenesis. Biological aspects of TSE diseases are reflected in the specificities of in vitro PrP conversion reactions. Here we show that there is a correlation between in vitro conversion efficiencies and known transmissibilities of BSE, sheep scrapie and CJD. On this basis, we used an in vitro system to gauge the potential transmissibility of scrapie and BSE to humans. We found limited conversion of human PrP-sen to PrP-res driven by PrP-res associated with both scrapie (PrP-Sc) and BSE (PrP-BSE). The efficiencies of these heterologous conversion reactions were similar but much lower than those of relevant homologous conversions. Thus the inherent ability of these infectious agents of BSE and scrapie to affect humans following equivalent exposure may be finite but similarly low.

L14 ANSWER 8 OF 9 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
DUPLICATE 6

AN 1996:526174 BIOSIS
DN PREV199699248530
TI PrP genotype contributes to determining survival times of sheep with natural scrapie.
AU ***Bossers, Alex*** ; Schreuder, Bram E. C.; Muileman, Ida H.; Belt, Peter B. G. M.; Smits, Mari A. [Reprint author]
CS Dep. Bacteriol., DLO-Inst. Anim. Sci. Health, P.O. Box 65, 8200 AB Lelystad, Netherlands
SO Journal of General Virology, (1996) Vol. 77, No. 10, pp. 2669-2673. CODEN: JGVIAY. ISSN: 0022-1317.
DT Article
LA English
ED Entered STN: 22 Nov 1996
Last Updated on STN: 22 Nov 1996
AB Several allelic variants of the sheep PrP gene are associated with scrapie susceptibility. However, it is not known whether, and to what extent, the PrP genotype contributes to determining survival times of scrapie sheep. We therefore determined the PrP genotype and life spans of over 50 Flemish and Swifter sheep within a single scrapie-affected flock. Eighty-three per cent of the scrapie sheep were homozygous for the PrPvQ allele (polymorphic amino acids at codons 136 and 171 are indicated) and these sheep died from scrapie at a mean age of 25 months. In sheep heterozygous for PrP-VQ, development of scrapie was delayed or did not occur. Sheep with at least one PrP-AR allele, including PrP-VQ/PrP-AR sheep, did not develop scrapie. No scrapie sheep were found without a PrP-VQ allele. We conclude that the PrP genotype contributes to determining survival times of sheep with natural scrapie. Additionally, we describe two novel sheep PrP allelic variants.

L14 ANSWER 9 OF 9 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1997:593179 CAPLUS
DN 127:246402
TI PrP allelic variants associated with natural scrapie
AU Belt, Peter B. G. M.; ***Bossers, Alex*** ; Schreuder, Bram E. C.; Smits, Mari A.
CS Department of Bacteriology, Institute for Animal Science and Health (ID-DLO), Lelystad, Neth.
SO Bovine Spongiform Encephalopathy: The BSE Dilemma, [Proceedings of the International Workshop on Bovine Spongiform Encephalopathy: The BSE Dilemma], 6th, Williamsburg, Va., Feb. 26-Mar. 1, 1995 (1996), 294-305. Editor(s): Gibbs, Clarence J. Publisher: Springer, New York, N. Y. CODEN: 64ZIAF
DT Conference
LA English
AB The PrP allelic variants of 69 scrapie-affected and 176 healthy sheep by denaturing gradient gel electrophoresis (DGGE) were detd. The results indicated that specific combinations of polymorphisms within the PrP gene of sheep, rather that single polymorphisms, are assocd. with the incidence of scrapie. A pos. selection for the PrPARR allele and/or a neg. selection for the PrPVRQ in breeding programs could help to control natural scrapie.

=> s prion? and (guanidine thiocyanate)
L15 103 PRION? AND (GUANIDINE THIOCYANATE)

=> dup rem l15
PROCESSING COMPLETED FOR L15
L16 47 DUP REM L15 (56 DUPLICATES REMOVED)

=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 47 ANSWERS - CONTINUE? Y/(N):y

L16 ANSWER 1 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2004:40993 CAPLUS
DN 140:90314
TI Sample preparation and immunoassay for the automated detection of proteinase resistant ***prion*** proteins (PrPres) on a solid surface with immobilized plasminogen
IN Morel, Nathalie; Creminon, Christophe; Grassi, Jacques
PA Commissariat A L'Energie Atomique, Fr.

SO Fr. Demande, 40 pp.
CODEN: FRXXBL
DT Patent
LA French
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI FR 2842303	A1	20040116	FR 2002-8608	20020709
WO 2004008144	A2	20040122	WO 2003-FR2117	20030708
WO 2004008144	A3	20040408		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI FR 2002-8608 A 20020709
AB The invention concerns an immunoassay for the automated detn. of proteinase resistant ***prion*** proteins (PrPres) on the surface of microtiterplates or magnetic spheres with immobilized plasminogen. The procedure includes: (a) sample prepn. (i) homogenization of the sample, e.g. sheep brain in a buffer contg. ionic and non-ionic surfactants, glucose, saccharose, phosphate and optionally proteinase K; (ii) treatment with a capture buffer that contains ionic surfactants and optionally proteinase K; (b) capturing the PrPres from the prepd. sample onto a surface with covalently immobilized plasminogen using the above buffer without proteinase K; (c) denaturation of PrPres on the plasminogen surface using a chaotropic agent at 100.degree.C; (d) detection of the denatured and immobilized PrPres using specific antibodies to protein PrP.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 2 OF 47 USPATFULL on STN
AN 2004:166069 USPATFULL
TI Sodium dodecyl sulfate compositions for inactivating ***prions***
IN Prusiner, Stanley B., San Francisco, CA, UNITED STATES
Supattapone, Surachai, Hanover, NH, UNITED STATES
PI US 2004127559 A1 20040701
AI US 2003-735454 A1 20031212 (10)
RLI Continuation of Ser. No. US 2002-56222, filed on 22 Jan 2002, GRANTED, Pat. No. US 6720355 Continuation-in-part of Ser. No. US 2001-904178, filed on 11 Jul 2001, GRANTED, Pat. No. US 6719988 Continuation-in-part of Ser. No. US 2000-699284, filed on 26 Oct 2000, ABANDONED Continuation-in-part of Ser. No. US 2000-494814, filed on 31 Jan 2000, GRANTED, Pat. No. US 6322802 Continuation-in-part of Ser. No. US 1999-447456, filed on 22 Nov 1999, GRANTED, Pat. No. US 6331296 Continuation-in-part of Ser. No. US 1999-322903, filed on 1 Jun 1999, GRANTED, Pat. No. US 6214366 Continuation-in-part of Ser. No. US 1999-235372, filed on 20 Jan 1999, GRANTED, Pat. No. US 6221614 Continuation-in-part of Ser. No. US 1998-151057, filed on 10 Sep 1998, ABANDONED Continuation-in-part of Ser. No. US 1998-26957, filed on 20 Feb 1998, ABANDONED Continuation-in-part of Ser. No. US 1997-804536, filed on 21 Feb 1997, GRANTED, Pat. No. US 5891641
DT Utility
FS APPLICATION
LREP BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD, SUITE 200, MENLO PARK, CA, 94025
CLMN Number of Claims: 41
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 3476
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An antiseptic composition useful in destroying the infectivity of infectious proteins such as ***prions*** is disclosed. The antiseptic composition is preferably maintained at either a low pH of 4.0 or less or a high pH of 10.0 or more either of which allows for an

environment under which the active component (which is preferably sodium dodecyl sulfate) destroys infectivity. The composition may be added to blood, blood products, collagen, tissues and organs prior to transplantation. The composition also may be added to livestock feed to denature any ***prions*** in the livestock. Methods of denaturing infectious proteins are also disclosed which method can use but do not require higher temperatures and long period of exposure.

L16 ANSWER 3 OF 47 USPATFULL on STN
AN 2004:166068 USPATFULL
TI Sodium dodecyl sulfate compositions for inactivating ***prions***
IN Prusiner, Stanley B., San Francisco, CA, UNITED STATES
Supattapone, Surachai, Hanover, NH, UNITED STATES
PA The Regents of the University of California (U.S. corporation)
PI US 2004127558 A1 20040701
AI US 2003-735140 A1 20031212 (10)
RLI Continuation of Ser. No. US 2002-56222, filed on 22 Jan 2002, GRANTED, Pat. No. US 6720355 Continuation-in-part of Ser. No. US 2001-904178, filed on 11 Jul 2001, GRANTED, Pat. No. US 6719988 Continuation-in-part of Ser. No. US 2000-699284, filed on 26 Oct 2000, ABANDONED Continuation-in-part of Ser. No. US 2000-494814, filed on 31 Jan 2000, GRANTED, Pat. No. US 6322802 Continuation-in-part of Ser. No. US 1999-447456, filed on 22 Nov 1999, GRANTED, Pat. No. US 6331296 Continuation-in-part of Ser. No. US 1999-322903, filed on 1 Jun 1999, GRANTED, Pat. No. US 6214366 Continuation-in-part of Ser. No. US 1999-235372, filed on 20 Jan 1999, GRANTED, Pat. No. US 6221614 Continuation-in-part of Ser. No. US 1998-151057, filed on 10 Sep 1998, ABANDONED Continuation-in-part of Ser. No. US 1998-26957, filed on 20 Feb 1998, ABANDONED Continuation-in-part of Ser. No. US 1997-804536, filed on 21 Feb 1997, GRANTED, Pat. No. US 5891641
DT Utility
FS APPLICATION
LREP BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD, SUITE 200, MENLO PARK, CA, 94025
CLMN Number of Claims: 38
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 3467
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An antiseptic composition useful in destroying the infectivity of infectious proteins such as ***prions*** is disclosed. The antiseptic composition is preferably maintained at either a low pH of 4.0 or less or a high pH of 10.0 or more either of which allows for an environment under which the active component (which is preferably sodium dodecyl sulfate) destroys infectivity. The composition may be added to blood, blood products, collagen, tissues and organs prior to transplantation. The composition also may be added to livestock feed to denature any ***prions*** in the livestock. Methods of denaturing infectious proteins are also disclosed which method can use but do not require higher temperatures and long period of exposure.

L16 ANSWER 4 OF 47 USPATFULL on STN
AN 2004:158591 USPATFULL
TI Method of preparing a standard diagnostic gene transcript pattern
IN Sharma, Praveen, Oslo, NORWAY
Lonneborg, Anders, Aas, NORWAY
PA DIAGENIC AS (non-U.S. corporation)
PI US 2004121390 A1 20040624
AI US 2003-727576 A1 20031205 (10)
RLI Division of Ser. No. US 1999-429003, filed on 29 Oct 1999, GRANTED, Pat. No. US 6720138 Continuation of Ser. No. WO 1998-GB1261, filed on 30 Apr 1998, UNKNOWN
PRAI NO 1997-2006 19970430
DT Utility
FS APPLICATION
LREP SUGHRUE MION, PLLC, 2100 Pennsylvania Avenue, N.W., Washington, DC, 20037-3213
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN 3 Drawing Page(s)
LN.CNT 1269

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for preparing a gene transcript pattern probe kit characteristic of a disease or condition or a stage thereof in a prokaryotic or eukaryotic organism using mRNA which is differentially expressed in the disease or condition or stage as probes, methods of diagnosis using the method and kits for performing the same are disclosed.

L16 ANSWER 5 OF 47 USPATFULL on STN
AN 2004:69606 USPATFULL
TI Sodium dodecyl sulfate compositions for inactivating ***prions***
IN Prusiner, Stanley B., San Francisco, CA, UNITED STATES
Supattapone, Surachai, Hanover, NH, UNITED STATES
PA The Regents of the University of California (U.S. corporation)
PI US 2004052833 A1 20040318
AI US 2003-641687 A1 20030814 (10)
RLI Continuation of Ser. No. US 2002-56222, filed on 22 Jan 2002, PENDING Continuation-in-part of Ser. No. US 2001-904178, filed on 11 Jul 2001, PENDING Continuation-in-part of Ser. No. US 2000-699284, filed on 26 Oct 2000, PENDING Continuation-in-part of Ser. No. US 2000-494814, filed on 31 Jan 2000, GRANTED, Pat. No. US 6322802 Continuation-in-part of Ser. No. US 1999-447456, filed on 22 Nov 1999, GRANTED, Pat. No. US 6331296 Continuation-in-part of Ser. No. US 1999-322903, filed on 1 Jun 1999, GRANTED, Pat. No. US 6214366 Continuation-in-part of Ser. No. US 1999-235372, filed on 20 Jan 1999, GRANTED, Pat. No. US 6221614 Continuation-in-part of Ser. No. US 1998-151057, filed on 10 Sep 1998, ABANDONED Continuation-in-part of Ser. No. US 1998-26957, filed on 20 Feb 1998, ABANDONED Continuation-in-part of Ser. No. US 1997-804536, filed on 21 Feb 1997, GRANTED, Pat. No. US 5891641
DT Utility
FS APPLICATION
LREP BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD, SUITE 200, MENLO PARK, CA, 94025
CLMN Number of Claims: 38
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 3478
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An antiseptic composition useful in destroying the infectivity of infectious proteins such as ***prions*** is disclosed. The antiseptic composition is preferably maintained at either a low pH of 4.0 or less or a high pH of 10.0 or more either of which allows for an environment under which the active component (which is preferably sodium dodecyl sulfate) destroys infectivity. The composition may be added to blood, blood products, collagen, tissues and organs prior to transplantation. The composition also may be added to livestock feed to denature any ***prions*** in the livestock. Methods of denaturing infectious proteins are also disclosed which method can use but do not require higher temperatures and long period of exposure.

L16 ANSWER 6 OF 47 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 1
AN 2004196462 EMBASE
TI [Creutzfeldt-Jakob disease and other human transmissible spongiform encephalopathies. Part II].
CHOROBA CREUTZFELDTA-JAKOBA I INNE PASAZOWALNE ENCEFALOPATIE GABCZASTE CZlOWIEKA. CZESC II.
AU Zaborowski A.
CS A. Zaborowski, Klin. Psychiat. Wieku Podeszl./Z. P., I Klinika Psychiatryczna, Uniwersytetu Medycznego, ul. Czechoslowacka 8/10, 92-216 Lodz, Poland
SO Psychiatria Polska, (2004) 38/2 (297-309).
Refs: 62
ISSN: 0033-2674 CODEN: PSPOB3
CY Poland
DT Journal; Article
FS 005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
032 Psychiatry
LA Polish
SL English; Bulgarian; German; French

AB The second part of this work presents the neuropathological problems of the Creutzfeldt-Jakob disease and basic informations about other human ***prion*** diseases. General problems of ***prion*** diseases and clinical symptoms of Creutzfeldt-Jakob disease were presented in the first part. ***Prion*** diseases are also known as transmissible cerebral amyloidoses (TCA) or transmissible (subacute) spongiform encephalopathies (TSE, SSE). There are following human TSE's: Creutzfeldt-Jakob disease (CJD) - the most frequent TSE, and its new variant (vCJD) - a result of BSE's transmission into human, sometimes treated as a aeparate disease; also: Gerstmann-Straussler-Scheinker syndrome (GSS) that may be a variant of familial CJD, kuru - probably a result of sporadic CJD's transmission by cannibalism, and fatal familial insomnia (FFI). Their clinical symptoms (and especially of the CJD), are nonspecific and sometimes variable. The imaging, EEG and other laboratory tests are not specific either. Neuropathological studies are needed but their interpretation may be equivocal. TSE's are characterised by the neurodegenerative process with characteristic spongiosis. However, vacuolisation - similar as in TSE-spongiosis - may occur in some CNS's disorders and in the case of putrescent brain tissue. In some cases of CJD, particularly those of long duration, the neuronal loss and astrocyte proliferation can mask the presence of spongiform changes, especially when vacuoles are not numerous. The only certain diagnostic marker for TSE is PrP(Sc), ***prion*** protein, presently believed to be a direct cause for all TSEs (TCAs). ThePrP(Sc) has a dominant .beta.-sheet amyloid structure which makes its detection by immunohistochemical procedure possible only with special pretreatment, e.c.: hydrolitic autoclaving, hydrated autoclaving, incubations: formic acid (or ***guanidine*** ***thiocyanate***) pretreatment, also combined pretreatments. These methods are standard diagnostic procedures for transmissible cerebral amyloidoses.

L16 ANSWER 7 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:349832 CAPLUS
DN 138:365135
TI Immunoassay reagent and kit for measuring abnormal-type ***prion*** , and immunoassay method for measuring abnormal-type ***prion*** using reagent or kit
IN Shinagawa, Shinichi; Horiuchi, Motohiro; Yanagitani, Takayuki; Matsui, Toshio; Umetani, Atsushi
PA Fujirebio, Inc., Japan
SO Jpn. Kokai Tokkyo Koho, 9 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2003130880	A2	20030508	JP 2001-330696	20011029
PRAI JP 2001-330696		20011029		

AB An immunoassay method is provided for detecting the abnormal-type ***prion*** with high sensitivity without performing a time-consuming electrophoresis operation or centrifugation operation. Also provided is an immunoassay reagent for this method, which is prepd. by immobilizing a first antibody immunol. reactive with the abnormal-type ***prion*** treated with a denaturing agent (e.g., guanidine, ***guanidine*** ***thiocyanate***) on magnetic particles. The method comprises a process for treating a sample potentially contg. the abnormal-type ***prion*** with a surfactant, collagenase and a proteinase (e.g., proteinase K), a process for treating the product obtained with a denaturing agent without having a centrifuge operation, and a process for immunol. assaying the product with the immunoassay reagent.

L16 ANSWER 8 OF 47 USPATFULL on STN
AN 2003:232025 USPATFULL
TI Ligands specific for an isoform of the ***prion*** protein
IN James, William Siward, Oxford, UNITED KINGDOM
Hope, James, Newbury, UNITED KINGDOM
Tahiri-Alaoui, Abdessamad, Oxford, UNITED KINGDOM
PI US 2003162225 A1 20030828
AI US 2002-295798 A1 20021115 (10)
RLI Continuation of Ser. No. WO 2001-GB2228, filed on 18 May 2001, UNKNOWN
PRAI GB 2000-12054 20000518

DT Utility
FS APPLICATION
LREP GRAY CARY WARE & FREIDENRICH LLP, 4365 EXECUTIVE DRIVE, SUITE 1100, SAN DIEGO, CA, 92121-2133
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 10 Drawing Page(s)
LN.CNT 1030
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB ***Prion*** protein, PrP, ligands are provided, especially protease resistant and nuclease resistant ligands. Ligands selective for isoforms such as PrP.sup.SC can be prepared. In a related aspect, the PrP ligands are used in diagnostic tests for PrP. The ligands also have potential for a role in the development of therapeutic methods for treatment of TSEs.

L16 ANSWER 9 OF 47 USPATFULL on STN
AN 2003:194526 USPATFULL
TI Muscle sample prepared for ***prion*** assay
IN Prusiner, Stanley B., San Francisco, CA, UNITED STATES
Bosque, Patrick, Denver, CO, UNITED STATES
PI US 2003134337 A1 20030717
AI US 2002-211942 A1 20020802 (10)
PRAI US 2002-351525P 20020122 (60)
US 2001-323903P 20010920 (60)
DT Utility
FS APPLICATION
LREP BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD, SUITE 200, MENLO PARK, CA, 94025
CLMN Number of Claims: 33
ECL Exemplary Claim: 1
DRWN 9 Drawing Page(s)
LN.CNT 1977
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of preparing a sample of muscle tissue and of assaying the prepared sample to determine the presence of ***prions*** in the sample is disclosed. The muscle tissue is homogenized and mixed with a complexing agent which forms a complex with a higher specific gravity than PrP.sup.Sc, the complexing agent or other components of the homogenate. Gravity is then used (e.g. ultra centrifugation) to concentrate the complex and the concentrate is assayed to detect ***prions*** . The muscle tissue is preferably extracted from a muscle or group of muscles such as hind limb muscle which have a higher or more preferably the highest concentration of ***prions*** as compared to other muscle in the mammal.

L16 ANSWER 10 OF 47 USPATFULL on STN
AN 2003:173177 USPATFULL
TI Capture compounds, collections thereof and methods for analyzing the proteome and complex compositions
IN Koster, Hubert, La Jolla, CA, UNITED STATES
Siddiqi, Suhaib, Oceanside, CA, UNITED STATES
Little, Daniel P., Winchester, MA, UNITED STATES
PI US 2003119021 A1 20030626
AI US 2002-197954 A1 20020716 (10)
PRAI US 2001-306019P 20010716 (60)
US 2001-314123P 20010821 (60)
US 2002-363433P 20020311 (60)
DT Utility
FS APPLICATION
LREP STEPHANIE SEIDMAN, HELLER EHRMAN WHITE & MCAULIFFE LLP, 7th FL., 4350 LA JOLLA VILLAGE DRIVE, SAN DIEGO, CA, 92122-1246
CLMN Number of Claims: 125
ECL Exemplary Claim: 1
DRWN 70 Drawing Page(s)
LN.CNT 6373
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Capture compounds and collections thereof and methods using the compounds for the analysis of biomolecules are provided. In particular, collections, compounds and methods are provided for analyzing complex protein mixtures, such as the proteome. The compounds are

multifunctional reagents that provide for the separation and isolation of complex protein mixtures. Automated systems for performing the methods also provided.

L16 ANSWER 11 OF 47 USPATFULL on STN
AN 2003:64747 USPATFULL
TI Method for detecting ***prion*** proteins in tissue samples
IN Aslamkhan, Abubakr, Durham, NC, UNITED STATES
Higgins, Donald, Franklinton, NC, UNITED STATES
PI US 2003044868 A1 20030306
AI US 2001-924812 A1 20010808 (9)
DT Utility
FS APPLICATION
LREP PARADIGM GENETICS, INC, 108 ALEXANDER DRIVE, P O BOX 14528, RTP, NC, 27709-4528
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN 4 Drawing Page(s)
LN.CNT 778
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Surprisingly, the present inventors have discovered that thermal denaturation of ***prion*** protein facilitates its detection by immunological methods. Accordingly, the present invention provides methods for the preparation and thermal denaturation of samples for ***prion*** detection, comprising: homogenizing a candidate sample and heating said sample in a buffer, preferably one with properties that aid stabilization of the denatured form of the protein. The methods described in this disclosure can be used in the detection of PrP.sup.Sc. Such detection is useful for the diagnosis of transmissible spongiform encephalopathies. This method can be used with immunoassays of various formats, including, but not limited to, dot blot and western blot assays, which utilize polyclonal antibodies, monoclonal antibodies, antibody fragments, receptors, natural and synthetic ligands and other entities.

L16 ANSWER 12 OF 47 USPATFULL on STN
AN 2003:30296 USPATFULL
TI Protein aggregation assays and uses thereof
IN Kondejewski, Les, St. Lazare, CANADA
Chakrabartty, Avijit, Vaughan, CANADA
Qi, Xiao-Fei, Toronto, CANADA
Cashman, Neil, Toronto, CANADA
PI US 2003022243 A1 20030130
AI US 2002-176809 A1 20020620 (10)
PRAI US 2001-299849P 20010620 (60)
DT Utility
FS APPLICATION
LREP CLARK & ELBING LLP, 101 FEDERAL STREET, BOSTON, MA, 02110
CLMN Number of Claims: 115
ECL Exemplary Claim: 1
DRWN 23 Drawing Page(s)
LN.CNT 2602
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention features methods for identifying agents that modulate protein aggregation or stabilize protein conformation. Exemplary methods include an in vitro aggregation assay, a native state stabilization assay, a cell-based screening assay, and an animal-based screening assay. These methods can be used to identify agents useful for the treatment of conformational diseases resulting from aggregation of a protein.

L16 ANSWER 13 OF 47 USPATFULL on STN
AN 2003:17028 USPATFULL
TI Polymer conjugates of proteinases
IN Sherman, Merry R., San Carlos, CA, UNITED STATES
Martinez, Alexa L., San Jose, CA, UNITED STATES
Bhaskaran, Shyam S., San Bruno, CA, UNITED STATES
Williams, L. David, Fremont, CA, UNITED STATES
Saifer, Mark G., San Carlos, CA, UNITED STATES
French, John A., Santa Cruz, CA, UNITED STATES
PI US 2003012777 A1 20030116

AI US 2002-183607 A1 20020628 (10)
RLI Continuation-in-part of Ser. No. US 2002-103128, filed on 22 Mar 2002, PENDING Continuation-in-part of Ser. No. US 2001-894071, filed on 28 Jun 2001, ABANDONED
DT Utility
FS APPLICATION
LREP STERNE, KESSLER, GOLDSTEIN & FOX PLLC, 1100 NEW YORK AVENUE, N.W., SUITE 600, WASHINGTON, DC, 20005-3934
CLMN Number of Claims: 143
ECL Exemplary Claim: 1
DRWN 18 Drawing Page(s)
LN.CNT 2195
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods are provided for the stabilization of proteinases by the covalent attachment of or admixture with water-soluble polymers. The resultant stabilized proteinases have increased stability under the harsh conditions used in industrial genomics, which permits their use in the extraction and isolation of nucleic acids and the identification of disease-related ***prion*** proteins at elevated temperatures in solutions containing chaotropic agents, such as sodium dodecyl sulfate, urea or guanidinium salts, conferring advantages for robotic applications.

L16 ANSWER 14 OF 47 USPATFULL on STN
AN 2003:4268 USPATFULL
TI Sodium dodecyl sulfate compositions for inactivating ***prions***
IN Prusiner, Stanley B., San Francisco, CA, UNITED STATES
Supattapone, Surachai, Hanover, NH, UNITED STATES
PI US 2003004312 A1 20030102
US 6720355 B2 20040413
AI US 2002-56222 A1 20020122 (10)
RLI Continuation-in-part of Ser. No. US 2001-904178, filed on 11 Jul 2001, PENDING Continuation-in-part of Ser. No. US 2000-699284, filed on 26 Oct 2000, PENDING Continuation-in-part of Ser. No. US 2000-494814, filed on 31 Jan 2000, GRANTED, Pat. No. US 6322802 Continuation-in-part of Ser. No. US 1999-447456, filed on 22 Nov 1999, GRANTED, Pat. No. US 6331296 Continuation-in-part of Ser. No. US 1999-322903, filed on 1 Jun 1999, GRANTED, Pat. No. US 6214366 Continuation-in-part of Ser. No. US 1999-235372, filed on 20 Jan 1999, GRANTED, Pat. No. US 6221614 Continuation-in-part of Ser. No. US 1998-151057, filed on 10 Sep 1998, ABANDONED Continuation-in-part of Ser. No. US 1998-26957, filed on 20 Feb 1998, ABANDONED Continuation-in-part of Ser. No. US 1997-804536, filed on 21 Feb 1997, GRANTED, Pat. No. US 5891641
DT Utility
FS APPLICATION
LREP BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD, SUITE 200, MENLO PARK, CA, 94025
CLMN Number of Claims: 38
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 3471
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An antiseptic composition useful in destroying the infectivity of infectious proteins such as ***prions*** is disclosed. The antiseptic composition is preferably maintained at either a low pH of 4.0 or less or a high pH of 10.0 or more either of which allows for an environment under which the active component (which is preferably sodium dodecyl sulfate) destroys infectivity. The composition may be added to blood, blood products, collagen, tissues and organs prior to transplantation. The composition also may be added to livestock feed to denature any ***prions*** in the livestock. Methods of denaturing infectious proteins are also disclosed which method can use but do not require higher temperatures and long period of exposure.

L16 ANSWER 15 OF 47 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 2
AN 2004:94985 BIOSIS
DN PREV200400095412
TI In vitro evaluation of the anti- ***prionic*** activity of newly synthesized congo red derivatives.
AU Poli, Giorgio [Reprint Author]; Ponti, Wilma; Carcassola, Gabriella;

Ceciliani, Fabrizio; Colombo, Laura; Dall'Ara, Paola; Gervasoni, Marco; Giannino, Maria Laura; Martino, Piera Anna; Pollera, Claudia; Villa, Stefania; Salmona, Mario
CS Dipartimento di Patologia Animale, Igiene e Sanita Pubblica Veterinaria, Sezione di Microbiologia e Immunologia, Via Celoria 10, 20133, Milano, Italy
giorgio.poli@unimi.it
SO Arzneimittel-Forschung, (2003) Vol. 53, No. 12, pp. 875-888. print.
ISSN: 0004-4172 (ISSN print).
DT Article
LA English
ED Entered STN: 11 Feb 2004
Last Updated on STN: 11 Feb 2004
AB "Transmissible Spongiform Encephalopathies" (TSE) are a group of degenerative progressive fatal disorders of the CNS, affecting both humans and animals. The main pathogenic event is the conversion of cellular ***prion*** protein from the normal, enzyme-sensitive (PrPsen), to the insoluble proteinase K-resistant isoform (PrPres). Since the new juvenile variant of Creutzfeldt-Jakob disease (vCJD) is probably due to the transmission of Bovine Spongiform Encephalopathy (BSE) ***prion*** protein to man, therapeutic and preventive compounds for animals and humans are urgently needed. Congo Red (benzidine-diazo-bis-1-naphthylamine-4-sulfonic acid sodium salt, CAS 573-58-0, CR), an azoic dye that inhibits amyloid deposition, and some newly synthesized derivatives, more lipophilic and less toxic, were tested for their anti- ***prionic*** activity, in different experimental models. Cell-free experiments using the synthetic peptide PrP 106-126, homologous to amino acid residues 106-126 of the human PrP, were run to determine the antiamyloidogenic properties of some of the molecules. Peptide solutions containing each compound were incubated at 37degreeC, for increasing times, to analyse the kinetics of aggregation of PrP 106-126 peptide. After incubation, the amount of non-aggregated peptide was measured by RP-HPLC. While CR enhanced the amyloidogenicity of PrP 106-126, derivatives "1a" and "1b" both showed the opposite behaviour, reducing aggregation by 15-20%. In other experiments using electron microscopy PrP 106-126 was assayed with the same molecules to assess the number and size of fibrils formed. CR showed its typical interaction, producing amyloid aggregates; "1a" did not interfere with fibril formation, while "1b" seemed to partially affect the structure of PrP 106-126 fibrils. Using a different cell-free model, it was investigated whether CR derivatives could reverse the protease-resistant PrPres, extracted from Syrian hamster infected brain, into the normal protease sensitive PrPsen. Samples containing fixed amounts of PrPres were incubated at 37degreeC for 1 h with all the newly synthesized molecules, at concentrations ranging from 50 mug/mL to 750 mug/mL. After treatment with proteinase K, half of each sample was incubated with 3 mol/L ***guanidine*** ***thiocyanate*** in order to exclude over-stabilisation of the PrPres aggregates already observed with CR. The remaining amount of PrPres was assessed by Enhanced Chemoluminescence (ECL) Western blotting analysis. None of the compounds induced the reversion of PrPres to PrPsen; nevertheless, 6 of the 8 molecules interacted with PrPres molecules, over-stabilising the PrPres aggregates, from this aspect being similar to CR in activity. Finally, the inhibition of the generation of PrPres in the S12 clone of a mouse neuroblastoma cell line (N2a S12), persistently infected by the mouse adapted Chandler strain of scrapie, was evaluated. Increasing amounts of CR, "1a" and "1b" were added to the culture medium at each cell passage. After various days of treatment, the cells were collected, lysed, and the amount of PrPres was assayed by ECL Western blotting after PK treatment. As expected, there was a decrease in pathological PrP expression starting from the 4th day of treatment, with 5 and 10 mug/mL CR; PrPres completely disappeared after respectively 10 and 14 days of treatment. "1a" was strongly effective after 3 days of treatment at 5 and 10 mug/mL, but it was also highly toxic; at the concentration of 1 mug/mL, it had a mild inhibitory effect after 8 days. The reduction of PrPres was also evaluated by intracytoplasmic flow-cytometry immunofluorescence on CR- and "1a"-treated N2a S12 cells. CR induced a dose-related decrease of PrP expression from day 3 to 13 of treatment. At the concentrations of 2 and 1.5 mug/mL "1a" also strongly affected the expression of PrP starting from the 3rd day of treatment until the end of the experiment (day 13). These results confirm the importance of using an integrated system, based on different experimental models, to obtain useful information on the

mechanism of action of anti- ***prionic*** compounds.

L16 ANSWER 16 OF 47 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 2003247281 EMBASE
TI ***Prions*** and orthopedic surgery.
AU Doerr H.W.; Cinatl J.; Sturmer M.; Rabenau H.F.
CS H.W. Doerr, Institute for Medical Virology, Johann Wolfgang Goethe University, Paul-Ehrlich-Str. 40, D-60596 Frankfurt/Main, Germany. H.W.Doerr@em.uni-frankfurt.de
SO Infection, (2003) 31/3 (163-171).
Refs: 39
ISSN: 0300-8126 CODEN: IFTNAL
CY Germany
DT Journal; General Review
FS 004 Microbiology
005 General Pathology and Pathological Anatomy
033 Orthopedic Surgery
LA English
SL English
AB ***Prions*** are a novel class of infectious agents that cause subacute encephalopathy in man and animals as human Creutzfeldt-Jakob disease (CJD), sheep scrapie and bovine spongiforrn encephalopathy (BSE). Previously, ***prions*** were shown to be transmitted by neuro- and ophthalmosurgical measures and by application of brain-derived therapeutic hormones. Recently, ***prions*** have been detected in blood specimens of experimentally infected monkeys indicating a principal threat to transfusion medicine, furthermore in human or bovine materials used in reconstitutive surgery. In this article the risk of ***prion*** transmission from the surgeon to the patient or vice versa during (orthopedic) surgery is reevaluated including the issues of blood transfusion. This is accomplished based on recent epidemiologic findings and biometric calculations on the spread of ***prions*** in animals and humans as well as in terms of experimental data on artificially contaminated medical materials and devices. The overall risk of ***prion*** transmission in orthopedic surgery is considered very low if adequately prepared and sterilized materials and devices are used.

L16 ANSWER 17 OF 47 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 3
AN 2003:374228 BIOSIS
DN PREV200300374228
TI Immunohistochemical investigations of the ***prion*** protein accumulation in human spongiform encephalopathies. Special report II.
AU Zaborowski, Adam; Kordek, Radzislaw; Botts, Gerald T.; Liberski, Pawel P. [Reprint Author]
CS Department of Molecular Pathology and Neuropathology, Chair of Oncology, Medical University, Czechoslowacka 8/10, 92-216, Lodz, Poland ppliber@csk.am.lodz.pl
SO Polish Journal of Pathology, (2003) Vol. 54, No. 1, pp. 39-47. print.
ISSN: 1233-9687.
DT Article
LA English
ED Entered STN: 13 Aug 2003
Last Updated on STN: 13 Aug 2003
AB Creutzfeldt-Jakob disease (CJD) in a proportion of cases may have nonspecific clinical signs and symptoms and no characteristic neuroimaging and EEG picture. Thus, neuropathological studies are mandatory for a diagnosis. However, spongiform change, neuronal loss and astrocyte proliferation - the hallmarks of ***prion*** diseases, may also be absent or variable. In such cases, the diagnosis should be supported by the detection of ***prion*** protein (PrP) by Western blotting or immunohistochemistry (ICC). PrP may not be visualised under "regular" conditions, but it is unmasked following pretreatment procedures: incubation in formic acid or ***guanidine*** ***thiocyanate*** , microwave treatment, and hydrated or hydrolytic autoclaving, and these methods were included in standard diagnostic procedures in several different protocols. The aim of this study was to compare the effectiveness of these pretreatment methods and to introduce an optimal protocol for our laboratory. For this purpose, we used brain sections of 11 cases of CJD, 1 case of Gerstmann-Straussler-Scheinker syndrome (GSS),

1 case of kuru and 3 control brains. For pretreatment we used the hydrated and hydrolytic autoclaving and incubation with formic acid. Immunostaining was performed with monoclonal 3F4 antibody against PrP. The best results were achieved with hydrolytic autoclaving. By this procedure we were able to detect the "synaptic" type of PrP accumulation in all CJD cases, as well as in GSS and kuru, while with other two methods the signal was weaker or even absent.

L16 ANSWER 18 OF 47 USPATFULL on STN
AN 2002:258862 USPATFULL
TI Human endosulfine gene
IN Roch, Jean-Marc, Waukegan, IL, UNITED STATES
Scott, Victoria E.S., Evanston, IL, UNITED STATES
Anderson, Kristi L., Grayslake, IL, UNITED STATES
Sullivan, James P., Deerfield, IL, UNITED STATES
PI US 2002142432 A1 20021003
AI US 2001-824178 A1 20010402 (9)
RLI Continuation of Ser. No. US 1997-779775, filed on 7 Jan 1997, ABANDONED
DT Utility
FS APPLICATION
LREP Steven F. Weinstock, Abbott Laboratories, Department 377 / AP6D-2, 100 Abbott Park Road, Abbott Park, IL, 60064-6050
CLMN Number of Claims: 40
ECL Exemplary Claim: 1
DRWN 8 Drawing Page(s)
LN.CNT 2951
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides an isolated or purified polynucleotide that encodes human endosulfine polypeptide. Isoforms of human endosulfine are also disclosed. The invention also provides methods of making recombinant human endosulfine using the polynucleotides and host cells transformed with the polynucleotides.

L16 ANSWER 19 OF 47 USPATFULL on STN
AN 2002:78206 USPATFULL
TI Antiseptic compositions for inactivating ***prions***
IN Prusiner, Stanley B., San Francisco, CA, UNITED STATES
Supattapone, Surachai, Hanover, NH, UNITED STATES
PI US 2002041859 A1 20020411
US 6719988 B2 20040413
AI US 2001-904178 A1 20010711 (9)
RLI Continuation-in-part of Ser. No. US 2000-699284, filed on 26 Oct 2000, PENDING Continuation-in-part of Ser. No. US 2000-494814, filed on 31 Jan 2000, GRANTED, Pat. No. US 6322802 Continuation-in-part of Ser. No. US 1999-447456, filed on 22 Nov 1999, PENDING Continuation-in-part of Ser. No. US 1999-322903, filed on 1 Jun 1999, GRANTED, Pat. No. US 6214366 Continuation-in-part of Ser. No. US 1999-235372, filed on 20 Jan 1999, GRANTED, Pat. No. US 6221614 Continuation-in-part of Ser. No. US 1998-151057, filed on 10 Sep 1998, ABANDONED Continuation-in-part of Ser. No. US 1998-26957, filed on 20 Feb 1998, ABANDONED Continuation-in-part of Ser. No. US 1997-804536, filed on 21 Feb 1997, GRANTED, Pat. No. US 5891641
DT Utility
FS APPLICATION
LREP Karl Bozicevic, Bozicevic, Field and Francis LLP, Suite 200, 200 Middlefield Road, Menlo Park, CA, 94025
CLMN Number of Claims: 22
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 3354
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An antiseptic composition useful in destroying the infectivity of infectious proteins such as ***prions*** is disclosed. The antiseptic composition is preferably maintained at a pH of 4.0 or less which allows for an environment under which the active component destroys infectivity. The composition may be added to blood, blood products, collagen, tissues and organs prior to transplantation. The composition also may be added to livestock feed to denature any ***prions*** in the livestock. Methods of denaturing infectious proteins are also disclosed.

L16 ANSWER 20 OF 47 USPATFULL on STN
AN 2002:37505 USPATFULL
TI METHOD OF PREPARING A STANDARD DIAGNOSTIC GENE TRANSCRIPT PATTERN
IN SHARMA, PRAVEEN, OSLO, NORWAY
LONNEBORG, ANDERS, AAS, NORWAY
PI US 2002022222 A1 20020221
US 6720138 B2 20040413
AI US 1999-429003 A1 19991029 (9)
RLI Continuation of Ser. No. WO 1998-GB1261, filed on 30 Apr 1998, UNKNOWN
PRAI NO 1997-2006 19970430
DT Utility
FS APPLICATION
LREP SUGHRUE MION ZINN MACPEAK & SEAS PLLC, 2100 PENNSYLVANIA AVENUE NW, WASHINGTON, DC, 200373213
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN 3 Drawing Page(s)
LN.CNT 1238
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for preparing a gene transcript pattern probe kit characteristic of a disease or condition or a stage thereof in a prokaryotic or eukaryotic organism using mRNA which is differentially expressed in the disease or condition or stage as probes, methods of diagnosis using the method and kits for performing the same are disclosed.

L16 ANSWER 21 OF 47 USPATFULL on STN
AN 2002:246898 USPATFULL
TI Transgenic mice expressing human APP and TGF-.beta. demonstrate cerebrovascular amyloid deposits
IN Mucke, Lennart, Foster City, CA, United States
Wyss-Coray, Tony, Berkeley, CA, United States
Masliah, Eliezer, Chula Vista, CA, United States
PA The Regents of the University of California, Oakland, CA, United States (U.S. corporation)
PI US 6455757 B1 20020924
AI US 1999-262519 19990304 (9)
RLI Continuation-in-part of Ser. No. US 1997-947295, filed on 8 Oct 1997
DT Utility
FS GRANTED
EXNAM Primary Examiner: Crouch, Deborah
LREP Francis, Carol L., Borden, Paula A., Bozicevic, Field & Francis, LLP
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 1966
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention features non-human transgenic animal models for Alzheimer's disease (AD) and CAA, wherein the transgenic animal is characterized by 1) expression of bioactive transforming growth factor-.beta.1 (TGF-.beta.1) or 2) both expression of bioactive TGF-.beta.1 and expression of a human amyloid .beta. precursor protein (APP) gene product. The transgenic animals may be either homozygous or heterozygous for these alterations. Bigenic animals are further characterized by development of AD-associated and/or CAA-associated pathology within about two to three months of age, and at about twelve months of age are characterized by a reduced number of neuritic plaques relative to singly transgenic animals. The invention also features methods of screening for biologically active agents that facilitate reduction of .beta.-amyloid deposits in vivo and methods for facilitating reduction of formation of neuritic plaques in a subject susceptible to AD.

L16 ANSWER 22 OF 47 USPATFULL on STN
AN 2002:152685 USPATFULL
TI Compositions and methods for advanced glycosylation endproduct-mediated modulation of amyloidosis
IN Vitek, Michael P., 205 Park Knoll La., Apex, NC, United States 27502
Cerami, Anthony, Ram Island Dr., Shelter Island, NY, United States 11964
Bucala, Richard J., 504 E. 63rd St. Apt. 33-0, New York, NY, United

States 10021
Ulrich, Peter C., 148 DeWolf Rd., Old Tappan, NJ, United States 07675
Vlassara, Helen, Ram Island Dr., Shelter Island, NY, United States 11964
Zhang, Xini, 150 Fairhaven Dr. Apt. D1, Jericho, NY, United States 117534)
PI US 6410598 B1 20020625
AI US 1995-477364 19950607 (8)
RLI Continuation-in-part of Ser. No. US 1995-457169, filed on 1 Jun 1995
Continuation-in-part of Ser. No. WO 1995-US1380, filed on 2 Feb 1995
Continuation-in-part of Ser. No. US 1994-311768, filed on 23 Sep 1994, now abandoned Continuation of Ser. No. US 1994-191579, filed on 3 Feb 1994, now abandoned
DT Utility
FS GRANTED
EXNAM Primary Examiner: Duffy, Patricia A.
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 12 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 2202
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates generally to the non-enzymatic glycosylation of amyloidogenic proteins and the consequent formation of advanced glycosylation endproducts (AGEs). It has been found that formation of AGE-amyloidogenic proteins can enhance amyloidosis. The invention further relates to compositions and methods for the prevention and treatment of amyloidosis associated with amyloid diseases, particularly neurodegenerative disease and Type II diabetes, and more particularly Alzheimer's disease. In a specific example, aggregation of an amyloidogenic peptide, .beta.AP, is enhanced by the glycosylation reaction of .beta.AP to form AGE-.beta.AP as defined herein. Accordingly, the invention extends to a method for modulating the in vivo aggregation of amyloid polypeptides and associated amyloidosis by controlling the formation and presence of AGE-amyloid polypeptide. A corresponding diagnostic utility comprises the measurement of the course and extent of amyloidosis by a measurement of the presence and amount of AGEs and particularly, AGE-amyloid. An assay is included that may use the AGE-amyloid polypeptide of the present invention to identify disease states characterized by the presence of AGE-amyloid. Additionally, such an assay can be utilized to monitor therapy and thus adjust a dosage regimen for a given disease state characterized by the presence of AGE-amyloid.

L16 ANSWER 23 OF 47 MEDLINE on STN
AN 2002621819 MEDLINE
DN PubMed ID: 12379130
TI Protease-sensitive scrapie ***prion*** protein in aggregates of heterogeneous sizes.
AU Tzaban Salit; Friedlander Gilgi; Schonberger Oshrat; Horonchik Lior; Yedidia Yifat; Shaked Gideon; Gabizon Ruth; Taraboulos Albert
CS Department of Molecular Biology, The Hebrew University-Hadassah Medical School, and Hadassah University Hospital, Jerusalem 91120, Israel.
SO Biochemistry, (2002 Oct 22) 41 (42) 12868-75.
Journal code: 0370623. ISSN: 0006-2960.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200212
ED Entered STN: 20021017
Last Updated on STN: 20021217
Entered Medline: 20021211
AB The pathological ***prion*** protein PrP(Sc) is the only known component of the infectious ***prion*** . In cells infected with ***prions*** , PrP(Sc) is formed posttranslationally by the refolding of the benign cell surface glycoprotein PrP(C) into an aberrant conformation. The two PrP isoforms possess very different properties, as PrP(Sc) has a protease-resistant core, forms very large amyloidic aggregates in detergents, and is only weakly immunoreactive in its native form. We now show that ***prion*** -infected rodent brains and cultured cells contain previously unrecognized protease-sensitive PrP(Sc) varieties. In

both ionic (Sarkosyl) and nonionic (n-octyl beta-D-glucopyranoside) detergents, the novel protease-sensitive PrP(Sc) species formed aggregates as small as 600 kDa, as measured by gel filtration. The denaturation dependence of PrP(Sc) immunoreactivity correlated with the size of the aggregate. The small PrP(Sc) aggregates described here are consistent with the previous demonstration of scrapie infectivity in brain fractions with a sedimentation coefficient as small as 40 S [Prusiner et al. (1980) J. Neurochem. 35, 574-582]. Our results demonstrate for the first time that ***prion*** -infected tissues contain protease-sensitive PrP(Sc) molecules that form low MW aggregates. Whether these new PrP(Sc) species play a role in the biogenesis or the pathogenesis of ***prions*** remains to be established.

L16 ANSWER 24 OF 47 USPATFULL on STN
AN 2001:90277 USPATFULL
TI METHODS FOR IN VITRO SUSCEPTIBILITY TESTING OF CHLAMYDIA
IN STRATTON, CHARLES W, NASHVILLE, TN, United States
MITCHELL, WILLIAM M, NASHVILLE, TN, United States
PI US 2001002421 A1 20010531
US 6258532 B2 20010710
AI US 1998-25176 A1 19980218 (9)
RLI Continuation-in-part of Ser. No. US 1997-911593, filed on 14 Aug 1997, ABANDONED
DT Utility
FS APPLICATION
LREP KAREN F. ELBING, CLARK AND ELBING, 176 FEDERAL STREET, BOSTON, MA, 02110
CLMN Number of Claims: 55
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 763
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods for determining the susceptibility of intracellular pathogens, particularly Chlamydia, to single or combination of test agents are described. The methods can be used for in vitro or in vivo evaluation of agents that can be used as therapeutic agents in the treatment/eradication of pathogen infection in general or to target a specific infected organ. Assays which utilize nucleic amplification techniques (e.g., PCR) to determine effectiveness of the agent(s) evaluated are also described.

L16 ANSWER 25 OF 47 USPATFULL on STN
AN 2001:8223 USPATFULL
TI Transgenic mouse model of alzheimer's disease and cerebral amyloid angiopathy
IN Mucke, Lennart, Foster City, CA, United States
Wyss-Coray, Tony, Berkeley, CA, United States
Masliah, Eliezer, Chula Vista, CA, United States
PA The Regents of the University of California, Oakland, CA, United States (U.S. corporation)
PI US 6175057 B1 20010116
AI US 1997-947295 19971008 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Crouch, Deborah
LREP Francis, Carol L., Borden, Paula A.Bozicevic, Field & Francis LLP
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 1697
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention features non-human transgenic animal models for Alzheimer's disease (AD) and CAA, wherein the transgenic animal is characterized by 1) overexpression of bioactive transforming growth factor-.beta.1 (TGF-.beta.1) or 2) both overexpression of bioactive TGF-.beta.1 and expression of a human amyloid .beta. precursor protein (APP) gene product. The transgenic animals may be either homozygous or heterozygous for these alterations. Bigenic animals are further characterized by development of AD-associated and/or CAA-associated pathology within about two to three months of age.

L16 ANSWER 26 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN

AN 2000:574024 CAPLUS
DN 133:174276
TI ***Prion*** test using ***guanidine*** ***thiocyanate*** for reducing false positive test results
IN Garssen, Gerrit Jan; Jacobs, Jorg Gunther; Langeveld, Joannes Pieter Maria; Smits, Marinus Adrianus; Van Keulen, Lucien Johannes Mattheus; Schreuder, Bram Edward Cornelis; Bossers, Alexander
PA Stichting Dienst Landbouwkundig Onderzoek, Neth.
SO PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000048003	A1	20000817	WO 2000-NL79	20000209
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1151305	A1	20011107	EP 2000-904139	20000209
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
PRAI EP 1999-200391	A	19990211		
WO 2000-NL79	W	20000209		

AB The invention is related to diagnostic methods for detecting transmissible spongiform encephalopathies (TSEs) such as BSE and scrapie and related disease in humans. The invention provides use of ***guanidine*** ***thiocyanate*** (gdnSCN) or a functional equiv. thereof for treating at least one sample derived from a mammal, including humans for reducing the risk of scoring a false-pos. test result in testing said sample for the presence or absence of aberrant ***prion*** protein.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 27 OF 47 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 4
AN 2000:117032 BIOSIS
DN PREV200000117032
TI Creutzfeldt-Jakob disease: Carnoy's fixative improves the immunohistochemistry of the proteinase K-resistant ***prion*** protein.
AU Giaccone, Giorgio [Reprint author]; Canciani, Barbara; Puoti, Gianfranco; Rossi, Giacomina; Goffredo, Donato; Lussich, Selina; Fociani, Paolo; Tagliavini, Fabrizio; Bugiani, Orso
CS Istituto Neurologico Carlo Besta, via Celoria 11, 20133, Milano, Ml, Italy
SO Brain Pathology, (Jan., 2000) Vol. 10, No. 1, pp. 31-37. print.
ISSN: 1015-6305.
DT Article
LA English
ED Entered STN: 29 Mar 2000
Last Updated on STN: 3 Jan 2002
AB The neuropathological diagnosis of Creutzfeldt-Jakob disease relies on the immunohistochemical demonstration of the proteinase-K resistant form of the ***prion*** protein (PrPres) in the brain tissue. The antigenicity of PrPres is strongly reduced by the formalin solution widely used to fix the tissue, thus the PrPres immunoreactivity is inconsistently detectable in formalin-fixed tissue. A better PrPres immunostaining can be obtained by using Carnoy's fixing solution, which is composed of ethanol, chloroform and acetic acid (6:3:1). PrPres can easily be extracted from Carnoy's-fixed, paraplast-embedded tissue. Accordingly, Carnoy's-fixed tissue can prior to immunolabeling be subjected to proteinase K and ***guanidine*** ***thiocyanate*** , which respectively eliminate the normal cellular form of ***prion*** protein and promote protein denaturation. In comparison with the best protocols for formalin-fixed tissue (i.e.-hydrolytic autoclaving or autoclaving in

distilled water followed by formic acid and ***guanidine*** ***thiocyanate***), PrPres immunostaining carried out on sections cut from Carnoy's-fixed, paraplast-embedded tissue blocks and subjected to proteinase K and ***guanidine*** ***thiocyanate*** , proved more successful to detect and map both diffuse and focal PrPres immunoreactivity, and to correlate the immunoreactivity pattern with MV polymorphism at PRNP codon 129 and PrPres banding and glycosylation pattern revealed by Western blot.

L16 ANSWER 28 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 5
AN 2000:538380 CAPLUS
DN 134:82972
TI PrP immunohistochemistry: different protocols, including a procedure for long formalin fixation, and a proposed schematic classification for deposits in sporadic Creutzfeldt-Jakob disease
AU Privat, Nicolas; Sazdovitch, Veronique; Seilhean, Danielle; Laplanche, Jean-Louis; Hauw, Jean-Jacques
CS Raymond Escourolle Neuropathology Laboratory, Paris VI University, Paris, 75651, Fr.
SO Microscopy Research and Technique (2000), 50(1), 26-31
CODEN: MRTEEO; ISSN: 1059-910X
PB Wiley-Liss, Inc.
DT Journal
LA English
AB The use of immunohistochem. on formalin-fixed and paraffin-embedded tissue has greatly improved the neuropathol. diagnosis of Creutzfeldt-Jakob disease and the other subacute spongiform encephalopathies in human and animals. Two pitfalls of this technique, however, currently exist: low sensitivity after long formalin fixation and difficulties in interpreting some images. Here we review the protocols currently in use for the pretreatment of sections allowing PrP detection by immunohistochem. In addn., a technique useful after long formalin fixation is reported: enzymic digestion with proteinase K (24.degree.C, 1/100 for 8 min) was employed in addn. to the usual autoclaving (121.degree.C for 10 min) followed by formic acid (99% for 5 min) and 4M ***guanidine*** ***thiocyanate*** (4.degree.C for 2 h). This allowed a substantial increase in the sensitivity of 3F4 immunohistochem. on paraffin-embedded tissue, esp. after prolonged formalin fixation. In addn., we suggest a simple method for classification of PrP immunolabelling in sporadic Creutzfeldt-Jakob disease that would allow easy comparisons.
RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 29 OF 47 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 2000:271928 BIOSIS
DN PREV200000271928
TI A rapid method for PCR detection of bovine materials in animal feedstuffs.
AU Wang, R.-F. [Reprint author]; Myers, M. J.; Campbell, W.; Cao, W.-W.; Paine, D.; Cerniglia, E.
CS Microbiology Division, National Center for Toxicological Research, US Food and Drug Administration (FDA), Jefferson, AR, 72079, USA
SO Molecular and Cellular Probes, (Feb., 2000) Vol. 14, No. 1, pp. 1-5. print.
ISSN: 0890-8508.
DT Article
LA English
ED Entered STN: 30 Jun 2000
Last Updated on STN: 5 Jan 2002
AB Rapid identification of bovine materials in animal feedstuffs is essential for effective control of a potential source of bovine spongiform encephalopathy. We have developed a rapid method for the detection of the presence of bovine materials in animal feeds. Animal feed samples were prepared by a Chelex-100 treatment method, then subjected to polymerase chain reaction (PCR) detection. The assay can be completed in 2 h including 30 min for sample preparation, 35-65 min for PCR cycling and 30 min for gel electrophoresis. This method is not only rapid, simple and consistent, but also avoids a hazardous waste disposal issue associated with a previously described ***guanidine*** ***thiocyanate*** (GuSCN) extraction-PCR method.

L16 ANSWER 30 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1999:97206 CAPLUS
DN 130:206996
TI Concentration and detection of pathogenic ***prion*** proteins by ELISA
IN Shinagawa, Shinichi; Horiuchi, Motohiro
PA Sangi Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 18 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 11032795	A2	19990209	JP 1997-193801	19970718

PRAI JP 1997-193801 19970718

AB Pathogenic ***prion*** proteins in substances derived from animal tissues are detected by ELISA involving the steps of (1) homogenizing the substances with surfactants and enzymes, (2) degrading the homogenates with degrading enzymes, (3) concg. the ***prion*** proteins from the homogenates, (4) dissolving the concs. in solvents, (5) adsorbing the ***prion*** proteins in the solns. on surfaces, and (6) coloring the ***prion*** proteins adsorbed. N-dodecyl-N,N-dimethyl-3-amino-1-propane sulfonate or tert-octylphenoxypolyethoxyethanol may be used as the surfactants. The process, for concg. the ***prion*** proteins, involving the steps (1), (2), and (3) is also claimed. PrPsc in brain and spleen tissues of scrapie-infected mice was concd. and detected according to the method with high sensitivity.

L16 ANSWER 31 OF 47 USPATFULL on STN
AN 1999:92643 USPATFULL
TI Compositions and methods for stimulating amyloid removal in amyloidogenic diseases using advanced glycosylation endproducts
IN Vitek, Michael P., East Norwich, NY, United States
Cerami, Anthony, Shelter Island, NY, United States
Bucala, Richard J., New York, NY, United States
Ulrich, Peter C., Old Tappan, NJ, United States
Vlassara, Helen, Shelter Island, NJ, United States
Zhang, Xini, Jericho, NJ, United States
PA The Picower Institute For Medical Research, Manhasset, NY, United States (U.S. corporation)
PI US 5935927 19990810
WO 9520979 19950810
AI US 1996-501127 19960810 (8)
WO 1995-US1380 19950202
19960810 PCT 371 date
19960810 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 1994-311768, filed on 23 Sep 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-191579, filed on 3 Feb 1994, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Duffy, Patricia A.
LREP Klauber & Jackson
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 12 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 2154
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates generally to methods and compositions for treating amyloidogenic diseases such as Alzheimer's disease and the development of type II diabetes, in which deposition of amyloid in organs such as the brain and pancreas interfere with neurological function and insulin release, respectively. The methods and compositions are directed toward increasing the activity of scavenger cells within the body at recognizing and removing amyloid deposits from affected tissues and organs. Scavenger cells may be targeted to amyloid deposits by means of spontaneously-occurring chemical modifications called advanced glycosylation endproducts (AGEs). Compositions are described which increase scavenger cell activity towards AGE-modified amyloid. Amyloid removal may also be enhanced by increasing AGE levels in amyloid

deposits within the body by administering AGE-modified amyloid targeting agents, which after becoming situated at sites containing amyloid, subsequently attract scavenger cells to degrade attendant amyloid. These methods and associated compositions result in a decrease in the extent of amyloid deposits in tissues, reducing the attendant pathology.

L16 ANSWER 32 OF 47 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 6
AN 2000:830 BIOSIS
DN PREV200000000830
TI Detection of bovine spongiform encephalopathy-specific PrPSc by treatment with heat and ***guanidine*** ***thiocyanate*** .
AU Meyer, Rudolf K. [Reprint author]; Oesch, Bruno; Fatzer, Rosmarie; Zurbriggen, Andreas; Vandevelde, Marc
CS Institute of Animal Neurology, University of Bern, Bremgartenstrasse 109a, CH-3012, Bern, Switzerland
SO Journal of Virology, (Nov., 1999) Vol. 73, No. 11, pp. 9386-9392. print.
CODEN: JOVIAM. ISSN: 0022-538X.
DT Article
LA English
ED Entered STN: 23 Dec 1999
Last Updated on STN: 31 Dec 2001
AB The conversion of a ubiquitous cellular protein (PrPC), an isoform of the ***prion*** protein (PrP), to the pathology-associated isoform PrPSc is one of the hallmarks of transmissible spongiform encephalopathies such as bovine spongiform encephalopathy (BSE). Accumulation of PrPSc has been used to diagnose BSE. Here we describe a quantitative enzyme-linked immunosorbent assay (ELISA) that involves antibodies against epitopes within the protease-resistant core of the PrP molecule to measure the amount of PrP in brain tissues from animals with BSE and normal controls. In native tissue preparations, little difference was found between the two groups. However, following treatment of the tissue with heat and ***guanidine*** ***thiocyanate*** (Gh treatment), the ELISA discriminated BSE-specific PrPSc from PrPC in bovine brain homogenates. PrPSc was identified by Western blot, centrifugation, and protease digestion experiments. It was thought that folding or complexing of PrPSc is most probably rev ersed by the Gh treatment, making hidden antigenic sites accessible. The digestion experiments also showed that protease-resistant PrP in BSE is more difficult to detect than that in hamster scrapie. While the concentration of PrPC in cattle is similar to that in hamsters, PrPSc sparse in comparison. The detection of PrPSc by a simple physicochemical treatment without the need for protease digestion, as described in this study, could be applied to develop a diagnostic assay to screen large numbers of samples.

L16 ANSWER 33 OF 47 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN DUPLICATE 7
AN 1999250855 EMBASE
TI [Safety measures in handling patients and laboratory samples infected with Creutzfeldt-Jakob disease].
VOORZORGSMAATREGELEN BIJ OMGANG MET PATIENTEN EN LABORATORIUMMONSTERS BESMET MET DE ZIEKTE VAN CREUTZFELDT-JAKOB.
AU Van Everbroeck B.; Pals P.; Cras P.
CS B. Van Everbroeck, Universitaire Instelling Antwerpen, Born Bunge Stichting, Laboratorium Neurobiologie, Universiteitsplein 1, 2610 Wilrijk, Belgium
SO Nederlands Tijdschrift voor Geneeskunde, (17 Jul 1999) 143/29 (1511-1514).
Refs: 24
ISSN: 0028-2162 CODEN: NETJAN
CY Netherlands
DT Journal; Article
FS 035 Occupational Health and Industrial Medicine
LA Dutch
SL English; Dutch
AB - Creutzfeldt-Jakob disease (CLD) is a transmissible subacute spongiform encephalopathy that invariably leads to death. The presumed causative agent, the ***prion*** protein, is highly resistant to inactivation and has a long incubation period. - Physical contact with CID patients (as in clinical care) entails no risk of transmission. - During procedures such as lumbar puncture where contact with infected material is possible, precautions are necessary. - Precautions are: the use of gloves, maximal

protection of people who come in contact with contaminated tissue (e.g. pathologist and histological laboratory worker) and transportation of samples in a closed and labelled container. - For laboratory research the tissue must be submerged in 92-98% formic acid for 1 hour. - All used materials and instruments must be decontaminated properly, using for instance NaOH, NaClO, ***guanidine*** ***thiocyanate*** or steam autoclaving. - If adequate precautions are taken contact with contaminated materials can be safe.

L16 ANSWER 34 OF 47 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 8
AN 2000:37694 BIOSIS
DN PREV200000037694
TI Antigen retrieval in ***prion*** protein immunohistochemistry.
AU Van Everbroeck, Bart; Pals, Philippe; Martin, Jean-Jacques; Cras, Patrick [Reprint author]
CS Born Bunge Foundation, Laboratory of Neurobiology, University of Antwerp, Universiteitsplein 1, B-2610, Wilrijk, Belgium
SO Journal of Histochemistry and Cytochemistry, (Nov., 1999) Vol. 47, No. 11, pp. 1465-1470. print.
CODEN: JHCYAS. ISSN: 0022-1554.
DT Article
LA English
ED Entered STN: 19 Jan 2000
Last Updated on STN: 31 Dec 2001
AB Transmissible spongiform encephalopathies are a group of neurodegenerative diseases occurring in both humans and animals and are most likely caused by ***prions*** . Neuropathological confirmation of the clinical diagnosis has been a problem because of the difficulty in epitope retrieval from formalin-fixed, paraffin-embedded brain specimens. Many different protocols for the detection of ***prions*** in brain tissue have been used. Thus far, picric and/or formic acid, steam autoclaving at 121C of sections, microwave treatment, and 4 M ***guanidine*** ***thiocyanate*** treatment have been suggested. The objective of our experiment was to obtain the standard pretreatment(s) resulting in optimal immunostaining. In the experiment, successive tissue slides of brain specimens of several Creutzfeldt-Jakob disease and control patients were stained using different combinations of pretreatments. Using densitometric analysis, several well-defined locations per section were examined and ***prion*** immunostaining was quantified. The results showed that autoclaving is necessary for antigen retrieval and cannot be substituted by microwave treatment. The best results were obtained when the following combination was used in the specified order: 15 min saturated picric acid, 10 min steam autoclaving at 121C, 5 min 88% formic acid, and 2 hr 4 M ***guanidine*** ***thiocyanate*** at 4C.

L16 ANSWER 35 OF 47 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 9
AN 1998:130434 BIOSIS
DN PREV199800130434
TI The anti- ***prion*** activity of congo red: Putative mechanism.
AU Caspi, Sigal; Halimi, Michele; Yanai, Anat; Sasson, Shmuel Ben; Taraboulos, Albert; Gabizon, Ruth [Reprint author]
CS Dep. Neurol., Hadassah Univ. Hosp., Jerusalem 91120, Israel
SO Journal of Biological Chemistry, (Feb. 6, 1998) Vol. 273, No. 6, pp. 3484-3489. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 20 Mar 1998
Last Updated on STN: 20 Mar 1998
AB PrPSc, an abnormal conformational isoform of the normal ***prion*** protein, PrPC, is the only known component of the ***prion*** , a proteinacious agent that causes fatal neurodegenerative disorders in humans and other animals. The hallmark properties of PrPSc are its insolubility in nondenaturing detergents and its resistance to digestion by proteases. Anions such as Congo red (CR) have been shown to reduce the accumulation of PrPSc in a neuroblastoma cell line permanently infected with ***prions*** as well as to delay disease onset in rodents when administrated prophylactically. The mechanism by which such anti- ***prion*** agents operate is unknown. We show here that in vitro

incubation with CR renders native PrPSc resistant to denaturation by boiling SDS. This resulted from PrPSc conformation, since neither the properties of PrPC nor those of predenatured PrPSc were changed by the addition of CR. CR-PrPSc could only be denatured by the. addition of acidic 3 m ***guanidine*** ***thiocyanate*** . Since in vitro conversion experiments have suggested that partial denaturation may be required for PrPSc to serve as template in the PrPC fwdarw PrPSc conversion, we propose that CR inhibits ***prion*** propogation by overstabilizing the conformation of PrPSc molecules.

L16 ANSWER 36 OF 47 MEDLINE on STN
AN 1998022906 MEDLINE
DN PubMed ID: 9356250
TI A conformational transition at the N terminus of the ***prion*** protein features in formation of the scrapie isoform.
AU Peretz D; Williamson R A; Matsunaga Y; Serban H; Pinilla C; Bastidas R B; Rozenshteyn R; James T L; Houghten R A; Cohen F E; Prusiner S B; Burton D R
CS Department of Neurology, School of Pharmacy, University of California, San Francisco, CA 94143, USA.
NC AG02132 (NIA)
NS14069 (NINDS)
NS22786 (NINDS)
+
SO Journal of molecular biology, (1997 Oct 31) 273 (3) 614-22.
Journal code: 2985088R. ISSN: 0022-2836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199801
ED Entered STN: 19980130
Last Updated on STN: 19980130
Entered Medline: 19980120
AB The scrapie ***prion*** protein (PrPSc) is formed from the cellular isoform (PrPC) by a post-translational process that involves a profound conformational change. Linear epitopes for recombinant antibody Fab fragments (Fabs) on PrPC and on the protease-resistant core of PrPSc, designated PrP 27-30, were identified using ELISA and immunoprecipitation. An epitope region at the C terminus was accessible in both PrPC and PrP 27-30; in contrast, epitopes towards the N-terminal region (residues 90 to 120) were accessible in PrPC but largely cryptic in PrP 27-30. Denaturation of PrP 27-30 exposed the epitopes of the N-terminal domain. We argue from our findings that the major conformational change underlying PrPSc formation occurs within the N-terminal segment of PrP 27-30.

L16 ANSWER 37 OF 47 BIOSIS on STN DUPLICATE 10
AN 1997:205726 BIOSIS
DN PREV199799504929
TI Sensitive enzyme-linked immunosorbent assay for detection of PrP-Sc in crude tissue extracts from scrapie-affected mice.
AU Grathwohl, Kai-Uwe D.; Horiuchi, Motohiro; Ishiguro, Naotaka; Shinagawa, Morikazu [Reprint author]
CS Dep. Vet. Public Health, Obihiro Univ. Agric. Vet. Med., W 2-11, Inada-cho, Obihiro, Hokkaido 080, Japan
SO Journal of Virological Methods, (1997) Vol. 64, No. 2, pp. 205-216.
CODEN: JVMEDH. ISSN: 0166-0934.
DT Article
LA English
ED Entered STN: 12 May 1997
Last Updated on STN: 12 May 1997
AB An enzyme-linked immunosorbent assay (ELISA) was developed that detects PrP-SC in crude extracts from brain and spleen tissue of scrapie-affected mice with high sensitivity and specificity. Brain tissue was homogenized in 8% Zwittergent 3-12 and 0.5% Sarkosyl. The homogenate was treated with collagenase and DNase I and then subjected to proteinase K digestion. Precipitates containing PrP-SC were obtained by ultracentrifugation. Spleen tissue was homogenized in 4% Triton X-100 and 0.5% Sarkosyl, and the homogenate was treated firstly with collagenase and DNase I, and

secondly with proteinase K. PrP-SC was then extracted with 6.25% Sarkosyl and precipitated through salting-out with NaCl and by ultracentrifugation. When PrP-SC was dissolved in 3-4 M ***guanidine*** ***thiocyanate*** and adsorbed to microliter plates, strong and specific reactions to the formation of antigen-antibody complexes could be detected by ELISA. The sensitivity of PrP-SC-detection for this ELISA, as measured by serial dilution of scrapie material in tissue homogenates from uninfected animals, was equal or higher than that attained by Western blot. This ELISA is more rapid than Western blot and seems to be more suitable for screening large numbers of animals. It also has potential application for the diagnosis of the transmissible spongiform encephalopathies.

L16 ANSWER 38 OF 47 LIFESCI COPYRIGHT 2004 CSA on STN DUPLICATE 11
AN 97:59013 LIFESCI
TI Decontamination of Creutzfeldt-Jakob disease and other transmissible agents
AU Manuelidis, L.
CS Yale Med. Sch., Sect. Neuropathology, 310 Cedar St., New Haven, CT 06510, USA
SO J. NEUROVIROL., (1997) vol. 3, no. 1, pp. 62-65.
ISSN: 1355-0284.
DT Journal
FS N3; V
LA English
SL English
AB The bovine spongiform encephalopathy (BSE) epidemic in cows, and the recent BSE-linked human infections, present new public health problems. More rigorous measures are needed to prevent additional transmissions. Tissue from established but undiagnosed human infections can contaminate medical supplies and instruments. We tested ***guanidine*** ***thiocyanate*** (GdnSCN) solutions and found them to be highly effective in disrupting the infectious agent, even in very complex tissues such as whole brain. It may be prudent now to use this reagent routinely in surgical and other relevant settings.

L16 ANSWER 39 OF 47 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 12
AN 1997:160635 BIOSIS
DN PREV199799459838
TI ***Prion*** protein immunocytochemistry - UK five centre consensus report.
AU Bell, J. E. [Reprint author]; Gentleman, S. M.; Ironside, J. W.; McCardle, L.; Lantos, P. L.; Doey, L.; Lowe, J.; Fergusson, J.; Luthert, P.; McQuaid, S.; Allen, I. V.
CS Univ. Neuropathol. Unit, Western Gen. Hosp., Crewe Road, Edinburgh EH4 2XU, UK
SO Neuropathology and Applied Neurobiology, (1997) Vol. 23, No. 1, pp. 26-35.
CODEN: NANEDL. ISSN: 0305-1846.
DT Article
LA English
ED Entered STN: 15 Apr 1997
Last Updated on STN: 15 Apr 1997
AB Creutzfeldt-Jakob disease (CJD) and other ***prion*** diseases are associated with the deposition of insoluble ***prion*** protein (PrP-CJD) in the central nervous system (CNS). Antibodies raised against PrP-CJD) also react with its precursor protein, a soluble form of PrP (PrP-C), which is widely distributed in the normal CNS. This crossreactivity has in the past raised doubts as to the specificity and diagnostic reliability of PrP immunolocalization, especially in familial cases which are atypical clinically and which lack characteristic pathology findings. Following an MRC-funded workshop which focused on this problem, a multicentre prospective study was set up to identify a reliable protocol for PrP-CJD immunocytochemistry. Five UK centres took part in this study and demonstrated consistent staining of plaques, vacuolar deposits in severe spongiform change, and perineuronal deposits using a variety of antibodies and enhancement procedures. A protocol using formic acid, ***guanidine*** ***thiocyanate*** , and hydrated autoclaving pre-treatment in conjunction with a monoclonal PrP-CJD antibody produced the clearest immunochemical results and is presented as the consensus UK recommendation for PrP-CJD immunocytochemical procedures.

L16 ANSWER 40 OF 47 USPATFULL on STN
AN 94:11236 USPATFULL
TI Method of treating the symptoms of Alzheimer's disease
IN Wagle, Sudhakar S., Mequon, WI, United States
Steinbach, Thomas, Houston, TX, United States
Lawyer, Carl H., Mequon, WI, United States
Hermann, William J., Sealy, TX, United States
Gawish, Ali A. S., Mequon, WI, United States
PA Kremers-Urban Company, Mequon, WI, United States (U.S. corporation)
PI US 5284664 19940208
AI US 1992-835029 19920205 (7)
RLI Continuation-in-part of Ser. No. US 1991-803844, filed on 4 Dec 1991 which is a continuation-in-part of Ser. No. US 1991-728267, filed on 11 Jul 1991, now abandoned which is a continuation of Ser. No. US 1988-228364, filed on 4 Aug 1988, now patented, Pat. No. US 5055296
DT Utility
FS Granted
EXNAM Primary Examiner: Robinson, Douglas W.; Assistant Examiner: Witz, Jean C.
LREP Tilton, Fallon, Lungmus & Chestnut
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 729
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A therapeutic method for treating Alzheimer's or related disease. The method comprises administering a therapeutically-effective amount of a mammalian liver extract, the extract being characterized by being heat stable, insoluble in acetone and soluble in water, peptide or peptide fragment selected from the groups consisting of Sequence Identification Numbers 1-9.

L16 ANSWER 41 OF 47 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 13
AN 1994:451350 BIOSIS
DN PREV199497464350
TI ***Prion*** protein immunocytochemistry: Reliable protocols for the investigation of Creutzfeldt-Jakob disease.
AU Hayward, P. A. R.; Bell, J. E.; Ironside, J. W. [Reprint author]
CS CJD Surveillance Unit, Neuropathology Lab., Univ. Dep. Pathology, Western General Hosp., Crewe Road, Edinburgh EH4 2XU, UK
SO Neuropathology and Applied Neurobiology, (1994) Vol. 20, No. 4, pp. 375-383.
CODEN: NANEDL. ISSN: 0305-1846.
DT Article
LA English
ED Entered STN: 24 Oct 1994
Last Updated on STN: 24 Oct 1994
AB Current criteria for the histological diagnosis of Creutzfeldt-Jakob disease (CJD) include features such as spongiform change, neuronal loss and reactive gliosis which are shared to a varying extent with other neurodegenerative disorders. Reliable visualization of ***prion*** protein (PrP) has substantial potential value in diagnostic practice and as a research tool, since accumulation of the disease-associated isoform of this protein is apparently specific for spongiform encephalopathies. A number of antisera against PrP have previously been employed in conjunction with a range of pre-treatments designed to optimize the specificity of immunostaining: such varied usage makes the comparison and interpretation of results difficult. This study was undertaken to identify optimal combinations of each of three PrP antisera and five pre-treatments designed to specifically demonstrate disease-specific PrP in a series of seven CJD cases, six cases of Alzheimer-type dementia and six non-demented control cases. Specific staining of amyloid plaques, spongiform neuropil, neurons and, occasionally, astrocytes was achieved in CJD cases. Alzheimer and control cases were unstained. Use of formic acid with ***guanidine*** ***thiocyanate*** , and hydrolytic autoclaving with IB3 and SP30 antisera proved most effective and can be recommended for future immunocytochemical studies. PrP immununocytochemistry revealed a greater extent of subcortical neural involvement than routine histological techniques in CJD: the relationship between classical neuropathology in CJD and PrP accumulation as revealed

by immunocytochemistry is not clear cut and requires further investigation. These findings may help to broaden our understanding of human spongiform encephalopathies, and have implications for diagnostic practices in neuropathology.

L16 ANSWER 42 OF 47 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 14
AN 1994:35631 BIOSIS
DN PREV199497048631
TI Absence of protease-resistant ***prion*** protein in dementia characterized by neuronal loss and status spongiosus.
AU Pollanen, M. S.; Bergeron, C. [Reprint author]; Weyer, L.
CS Cent. Res. Neurodegenerative Dis., Tanz Neurosci. Build., Room 121, Univ. Toronto, 6 Queen's Park Cres. West, Toronto, ON M5S 1A8, Canada
SO Acta Neuropathologica, (1993) Vol. 86, No. 5, pp. 515-517.
CODEN: ANPTAL. ISSN: 0001-6322.
DT Article
LA English
ED Entered STN: 27 Jan 1994
Last Updated on STN: 27 Jan 1994
AB Dementia characterized by neuronal loss and status spongiosus (DNLS) is a non-Alzheimer degenerative process which is characterized by Pick-like lobar atrophy with neuronal depletion and gliosis of the cerebral cortex, corpus striatum, medial thalamus, and substantia nigra and the absence of neuronal inclusions. To further investigate the cause and pathogenesis of DNLS, we probed cerebral homogenates from three cases of DNLS for protease-resistant ***prion*** protein to determine if DNLS could be a variant of a human ***prion*** disease. Limited proteolysis of ***prion*** proteins and ***guanidine*** ***thiocyanate*** treatment of cortical homogenates was used to enrich potential abnormal ***prion*** protein immunoreactivity. Although protease-resistant ***prion*** protein was detected in a case of sporadic Creutzfeldt-Jakob disease no abnormal ***prion*** protein was found in the cases of DNLS. We conclude that DNLS is not a human ***prion*** disease and remains an important dementia of uncertain etiology.

L16 ANSWER 43 OF 47 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 15
AN 1992:120514 BIOSIS
DN PREV199293066314; BA93:66314
TI ULTRASTRUCTURAL LOCALIZATION OF SCRAPIE ***PRION*** PROTEINS IN CYTOPLASMIC VESICLES OF INFECTED CULTURED CELLS.
AU MCKINLEY M P [Reprint author]; TARABOULOS A; KENAGA L; SERBAN D; STIEBER A; DEARMOND S J; PRUSINER S B; GONATAS N
CS DEP NEUROLOGY, HSE-781, UNIV CALIFORNIA, SAN FRANCISCO, CA 94143-00518, USA
SO Laboratory Investigation, (1991) Vol. 65, No. 6, pp. 622-630.
CODEN: LAINAW. ISSN: 0023-6837.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 1 Mar 1992
Last Updated on STN: 1 Mar 1992
AB Infectious scrapie ***prions*** are composed largely, if not entirely, of an abnormal isoform of the ***prion*** protein (PrP) designated PrPSc. In scrapie-infected mouse neuroblastoma (ScN2a) and hamster brain (ScHaB) cells, PrPSc accumulates primarily within the cell cytoplasm, whereas cellular PrP (PrPC) is anchored to the external surface of the plasma membrane by a glycoinositol phospholipid moiety. To determine the subcellular localization of PrPSc, scrapie-infected cells were grown to .apprx. 75% confluency, fixed briefly, and then incubated with ***guanidine*** ***thiocyanate*** before antibody staining and examination by electron microscopy. PrPSc immunoreactivity was enhanced by denaturation with guanidine isothiocyanate which also permeabilized cells (Taraboulos et al., J Cell Biol 110:2117, 1990). As judged both by deposition of immunoperoxidase reaction product (diaminobenzidine) and by presence of immunogold particles, PrPSc was identified in discrete vesicular foci and some large bodies in the cytoplasm of scrapie-infected cells. Some vesicles with PrPSc staining also contained myelin figures resembling those found in autophagic vacuoles forming secondary lysosomes. The presence of PrPSc in secondary lysosomes is inferred from

colocalization of guanidine isothiocyanate enhanced PrP immunoreactivity and acid phosphatase. Neither the diaminobenzidine reaction product nor immunogold particles were observed in association with the nucleus, endoplasmic reticulum, or Golgi stacks. Exposure of scrapie-infected cells to the brefeldin A dispersed the Golgi apparatus but did not alter the morphologic distribution of PrPSc, indicating that no detectable PrPSc was associated with Golgi stacks. It remains to be established whether secondary lysosomes are involved in the post-translational formation of PrPSc.

L16 ANSWER 44 OF 47 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 16
AN 1992:6142 BIOSIS
DN PREV199293006142; BA93:6142
TI IMMUNOREACTIVITY OF CEREBRAL AMYLOIDOSIS IS ENHANCED BY PROTEIN DENATURATION TREATMENTS.
AU DOI-YI R [Reprint author]; KITAMOTO T; TATEISHI J
CS DEP NEUROPATHOL, NEUROL INST, FAC MED, KYUSHU UNIV, 3-1-1 MAIDASHI, HIGASHIKU, FUKUOKA 812, JAPAN
SO Acta Neuropathologica, (1991) Vol. 82, No. 4, pp. 260-265.
CODEN: ANPTAL. ISSN: 0001-6322.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 10 Dec 1991
Last Updated on STN: 6 Mar 1992
AB We investigated paraffin-embedded brain sections from three patients with Gerstmann-Straussler syndrome and three patients with Alzheimer's disease or senile dementia of Alzheimer type using anti-human ***prion*** protein antisera and anti-.beta./A4 protein antisera after protein denaturation treatments. After incubation with ***guanidine*** - ***thiocyanate*** , trichloroacetate, and phenol, the immunoreactivity of kuru plaques and senile plaques was enhanced to the same level as the formic acid treatment. These treatments revealed small compact amyloid deposits, amyloid deposits surrounding the plaque cores, and diffuse plaques. Most of these chemicals changed the congophilia of both amyloids. It is possible that these treatments denature amyloid fibril proteins and break down the structure of amyloid fibrils, thus revealing buried epitopes.

L16 ANSWER 45 OF 47 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
AN 91:543273 SCISEARCH
GA The Genuine Article (R) Number: GG487
TI IMMUNOREACTIVITY OF CEREBRAL AMYLOIDOSIS IS ENHANCED BY PROTEIN DENATURATION TREATMENTS
AU DOIYI R (Reprint); KITAMOTO T; TATEISHI J
CS KYUSHU UNIV, DIV MED, INST NEUROL, DEPT NEUROPATHOL, 3-1-1 MAIDASHI, HIGASHI KU, FUKUOKA 812, JAPAN (Reprint)
CYA JAPAN
SO ACTA NEUROPATHOLOGICA, (1991) Vol. 82, No. 4, pp. 260-265.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 32
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB We investigated paraffin-embedded brain sections from three patients with Gerstmann-Straussler syndrome and three patients with Alzheimer's disease or senile dementia of Alzheimer type using anti-human ***prion*** protein antisera and anti-beta/A4 protein antisera after protein denaturation treatments. After incubation with ***guanidine*** - ***thiocyanate*** , trichloroacetate, and phenol, the immunoreactivity of kuru plaques and senile plaques was enhanced to the same level as the formic acid treatment. These treatments revealed small compact amyloid deposits, amyloid deposits surrounding the plaque cores, and diffuse plaques. Most of these chemicals changed the congophilia of both amyloids. It is possible that these treatments denature amyloid fibril proteins and break down the structure of amyloid fibrils, thus revealing buried epitopes.

L16 ANSWER 46 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN

AN 1991:614815 CAPLUS
DN 115:214815
TI Practical methods for chemical inactivation of the Creutzfeldt-Jakob disease pathogen
AU Tateishi, Jun; Tashma, Takatoshi; Kitamoto, Tetsuyuki
CS Fac. Med., Kyushu Univ., Fukuoka, 812, Japan
SO Microbiology and Immunology (1991), 35(2), 163-6
CODEN: MIIMDV; ISSN: 0385-5600
DT Journal
LA English
AB Chem. inactivation of the pathogen of Creutzfeldt-Jakob disease (CJD) was examd. using the mouse-adapted CJD strain. A high concn. of formic acid, guanidine compds., trichloroacetate and phenol prevented CJD transmission. NaOH between 0.25 and 2 N lengthened the incubation periods. Na dodecyl sulfate (SDS) in a concn. between 1 and 3% did not alter incubation at room temp. but did completely block the transmission after boiling for 3 min in 3% SDS. This method is recommended for practical disinfection.

L16 ANSWER 47 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1987:65309 CAPLUS
DN 106:65309
TI Congophilia in cerebral amyloidosis is modified by inactivation procedures on slow transmissible pathogens
AU Tashima, Takatoshi; Kitamoto, Tetsuyuki; Tateishi, Jun; Sato, Yuji
CS Fac. Med., Kyushu Univ., Fukuoka, 812, Japan
SO Brain Research (1986), 399(1), 80-6
CODEN: BRREAP; ISSN: 0006-8993
DT Journal
LA English
AB Cerebral tissues with amyloid deposits were treated by various chems. which inactivated the agent of subacute spongiform encephalopathy (SSE). Congophilia (affinity for Congo red) in the amyloid plaques in cases of Creutzfeldt-Jakob disease (CJD) and Gerstmann-Straussler syndrome (GSS) correlated to the chem. inactivation profiles of SSE. After incubation with trichloroacetate, guanidine-SCN, guanidine-HCl, formic acid, or phenol with autoclaving, amyloid plaques in unfixed frozen sections of human brains with CJD or GSS lost their affinity for Congo red and green birefringence under polarized light. In formalin-fixed, paraffin-embedded tissue sections, amyloid plaques of CJD and GSS lost their affinity for Congo red after most of these treatments. On the other hand, senile plaques in aged patients with Alzheimer's disease and with senile dementia of the Alzheimer type did not lose affinity for Congo red after most of these treatments. Therefore, the amyloid deposits in the amyloid plaques differ from those in senile plaques. These methods facilitate differentiation of amyloid and senile plaques in formalin-fixed, paraffin-embedded tissues.

=> logoff y
STN INTERNATIONAL LOGOFF AT 17:24:46 ON 07 SEP 2004

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* * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * * *
FILE 'HOME' ENTERED AT 17:25:08 ON 07 SEP 2004
=> file uspatfull
=> s prion? and (guanidine thiocyanate?)
          2960 PRION?
         26378 GUANIDINE
         25478 THIOCYANATE?
          1590 GUANIDINE THIOCYANATE?
                 (GUANIDINE(W)THIOCYANATE?)
L1          20 PRION? AND (GUANIDINE THIOCYANATE?)
=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 20 ANSWERS - CONTINUE? Y/(N):y
 L1   ANSWER 1 OF 20  USPATFULL on STN
AN     2004:166069  USPATFULL
TI     Sodium dodecyl sulfate compositions for inactivating   ***prions***
IN     Prusiner, Stanley B., San Francisco, CA, UNITED STATES
       Supattapone, Surachai, Hanover, NH, UNITED STATES
PI     US 2004127559      A1   20040701
AI     US 2003-735454     A1   20031212 (10)
RLI    Continuation of Ser. No. US 2002-56222, filed on 22 Jan 2002, GRANTED,
       Pat. No. US 6720355 Continuation-in-part of Ser. No. US 2001-904178,
       filed on 11 Jul 2001, GRANTED, Pat. No. US 6719988 Continuation-in-part
       of Ser. No. US 2000-699284, filed on 26 Oct 2000, ABANDONED
       Continuation-in-part of Ser. No. US 2000-494814, filed on 31 Jan 2000,
       GRANTED, Pat. No. US 6322802 Continuation-in-part of Ser. No. US
       1999-447456, filed on 22 Nov 1999, GRANTED, Pat. No. US 6331296
       Continuation-in-part of Ser. No. US 1999-322903, filed on 1 Jun 1999,
       GRANTED, Pat. No. US 6214366 Continuation-in-part of Ser. No. US
       1999-235372, filed on 20 Jan 1999, GRANTED, Pat. No. US 6221614
       Continuation-in-part of Ser. No. US 1998-151057, filed on 10 Sep 1998,
       ABANDONED Continuation-in-part of Ser. No. US 1998-26957, filed on 20
       Feb 1998, ABANDONED Continuation-in-part of Ser. No. US 1997-804536,
       filed on 21 Feb 1997, GRANTED, Pat. No. US 5891641
DT     Utility
FS     APPLICATION
LREP   BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD, SUITE 200, MENLO
       PARK, CA, 94025
CLMN   Number of Claims: 41
ECL    Exemplary Claim: 1
DRWN   12 Drawing Page(s)
LN.CNT 3476
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB     An antiseptic composition useful in destroying the infectivity of
       infectious proteins such as   ***prions***   is disclosed. The
       antiseptic composition is preferably maintained at either a low pH of
       4.0 or less or a high pH of 10.0 or more either of which allows for an
       environment under which the active component (which is preferably sodium
       dodecyl sulfate) destroys infectivity. The composition may be added to
       blood, blood products, collagen, tissues and organs prior to
       transplantation. The composition also may be added to livestock feed to
       denature any   ***prions***   in the livestock. Methods of denaturing
       infectious proteins are also disclosed which method can use but do not
       require higher temperatures and long period of exposure.

L1   ANSWER 2 OF 20  USPATFULL on STN
AN     2004:166068  USPATFULL
TI     Sodium dodecyl sulfate compositions for inactivating   ***prions***
IN     Prusiner, Stanley B., San Francisco, CA, UNITED STATES
       Supattapone, Surachai, Hanover, NH, UNITED STATES
PA     The Regents of the University of California (U.S. corporation)
PI     US 2004127558      A1   20040701
AI     US 2003-735140     A1   20031212 (10)
RLI    Continuation of Ser. No. US 2002-56222, filed on 22 Jan 2002, GRANTED,
       Pat. No. US 6720355 Continuation-in-part of Ser. No. US 2001-904178,
       filed on 11 Jul 2001, GRANTED, Pat. No. US 6719988 Continuation-in-part
       of Ser. No. US 2000-699284, filed on 26 Oct 2000, ABANDONED
       Continuation-in-part of Ser. No. US 2000-494814, filed on 31 Jan 2000,
       GRANTED, Pat. No. US 6322802 Continuation-in-part of Ser. No. US
       1999-447456, filed on 22 Nov 1999, GRANTED, Pat. No. US 6331296
       Continuation-in-part of Ser. No. US 1999-322903, filed on 1 Jun 1999,
       GRANTED, Pat. No. US 6214366 Continuation-in-part of Ser. No. US
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1999-235372, filed on 20 Jan 1999, GRANTED, Pat. No. US 6221614 Continuation-in-part of Ser. No. US 1998-151057, filed on 10 Sep 1998, ABANDONED Continuation-in-part of Ser. No. US 1998-26957, filed on 20 Feb 1998, ABANDONED Continuation-in-part of Ser. No. US 1997-804536, filed on 21 Feb 1997, GRANTED, Pat. No. US 5891641

DT Utility

FS APPLICATION

LREP BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD, SUITE 200, MENLO PARK, CA, 94025

CLMN Number of Claims: 38

ECL Exemplary Claim: 1

DRWN 12 Drawing Page(s)

LN.CNT 3467

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB An antiseptic composition useful in destroying the infectivity of infectious proteins such as ***prions*** is disclosed. The antiseptic composition is preferably maintained at either a low pH of 4.0 or less or a high pH of 10.0 or more either of which allows for an environment under which the active component (which is preferably sodium dodecyl sulfate) destroys infectivity. The composition may be added to blood, blood products, collagen, tissues and organs prior to transplantation. The composition also may be added to livestock feed to denature any ***prions*** in the livestock. Methods of denaturing infectious proteins are also disclosed which method can use but do not require higher temperatures and long period of exposure.

L1 ANSWER 3 OF 20 USPATFULL on STN

AN 2004:158591 USPATFULL

TI Method of preparing a standard diagnostic gene transcript pattern

IN Sharma, Praveen, Oslo, NORWAY
Lonneborg, Anders, Aas, NORWAY

PA DIAGENIC AS (non-U.S. corporation)

PI US 2004121390 A1 20040624

AI US 2003-727576 A1 20031205 (10)

RLI Division of Ser. No. US 1999-429003, filed on 29 Oct 1999, GRANTED, Pat. No. US 6720138 Continuation of Ser. No. WO 1998-GB1261, filed on 30 Apr 1998, UNKNOWN

PRAI NO 1997-2006 19970430

DT Utility

FS APPLICATION

LREP SUGHRUE MION, PLLC, 2100 Pennsylvania Avenue, N.W., Washington, DC, 20037-3213

CLMN Number of Claims: 17

ECL Exemplary Claim: 1

DRWN 3 Drawing Page(s)

LN.CNT 1269

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A method for preparing a gene transcript pattern probe kit characteristic of a disease or condition or a stage thereof in a prokaryotic or eukaryotic organism using mRNA which is differentially expressed in the disease or condition or stage as probes, methods of diagnosis using the method and kits for performing the same are disclosed.

L1 ANSWER 4 OF 20 USPATFULL on STN

AN 2004:69606 USPATFULL

TI Sodium dodecyl sulfate compositions for inactivating ***prions***

IN Prusiner, Stanley B., San Francisco, CA, UNITED STATES
Supattapone, Surachai, Hanover, NH, UNITED STATES

PA The Regents of the University of California (U.S. corporation)

PI US 2004052833 A1 20040318

AI US 2003-641687 A1 20030814 (10)

RLI Continuation of Ser. No. US 2002-56222, filed on 22 Jan 2002, PENDING Continuation-in-part of Ser. No. US 2001-904178, filed on 11 Jul 2001, PENDING Continuation-in-part of Ser. No. US 2000-699284, filed on 26 Oct 2000, PENDING Continuation-in-part of Ser. No. US 2000-494814, filed on 31 Jan 2000, GRANTED, Pat. No. US 6322802 Continuation-in-part of Ser. No. US 1999-447456, filed on 22 Nov 1999, GRANTED, Pat. No. US 6331296 Continuation-in-part of Ser. No. US 1999-322903, filed on 1 Jun 1999, GRANTED, Pat. No. US 6214366 Continuation-in-part of Ser. No. US 1999-235372, filed on 20 Jan 1999, GRANTED, Pat. No. US 6221614

Continuation-in-part of Ser. No. US 1998-151057, filed on 10 Sep 1998, ABANDONED Continuation-in-part of Ser. No. US 1998-26957, filed on 20 Feb 1998, ABANDONED Continuation-in-part of Ser. No. US 1997-804536, filed on 21 Feb 1997, GRANTED, Pat. No. US 5891641
DT Utility
FS APPLICATION
LREP BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD, SUITE 200, MENLO PARK, CA, 94025
CLMN Number of Claims: 38
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 3478
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An antiseptic composition useful in destroying the infectivity of infectious proteins such as ***prions*** is disclosed. The antiseptic composition is preferably maintained at either a low pH of 4.0 or less or a high pH of 10.0 or more either of which allows for an environment under which the active component (which is preferably sodium dodecyl sulfate) destroys infectivity. The composition may be added to blood, blood products, collagen, tissues and organs prior to transplantation. The composition also may be added to livestock feed to denature any ***prions*** in the livestock. Methods of denaturing infectious proteins are also disclosed which method can use but do not require higher temperatures and long period of exposure.

L1 ANSWER 5 OF 20 USPATFULL on STN
AN 2003:232025 USPATFULL
TI Ligands specific for an isoform of the ***prion*** protein
IN James, William Siward, Oxford, UNITED KINGDOM
Hope, James, Newbury, UNITED KINGDOM
Tahiri-Alaoui, Abdessamad, Oxford, UNITED KINGDOM
PI US 2003162225 A1 20030828
AI US 2002-295798 A1 20021115 (10)
RLI Continuation of Ser. No. WO 2001-GB2228, filed on 18 May 2001, UNKNOWN
PRAI GB 2000-12054 20000518
DT Utility
FS APPLICATION
LREP GRAY CARY WARE & FREIDENRICH LLP, 4365 EXECUTIVE DRIVE, SUITE 1100, SAN DIEGO, CA, 92121-2133
CLMN Number of Claims: 10
ECL Exemplary Claim: 1
DRWN 10 Drawing Page(s)
LN.CNT 1030
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB ***Prion*** protein, PrP, ligands are provided, especially protease resistant and nuclease resistant ligands. Ligands selective for isoforms such as PrP.sup.SC can be prepared. In a related aspect, the PrP ligands are used in diagnostic tests for PrP. The ligands also have potential for a role in the development of therapeutic methods for treatment of TSEs.

L1 ANSWER 6 OF 20 USPATFULL on STN
AN 2003:194526 USPATFULL
TI Muscle sample prepared for ***prion*** assay
IN Prusiner, Stanley B., San Francisco, CA, UNITED STATES
Bosque, Patrick, Denver, CO, UNITED STATES
PI US 2003134337 A1 20030717
AI US 2002-211942 A1 20020802 (10)
PRAI US 2002-351525P 20020122 (60)
US 2001-323903P 20010920 (60)
DT Utility
FS APPLICATION
LREP BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD, SUITE 200, MENLO PARK, CA, 94025
CLMN Number of Claims: 33
ECL Exemplary Claim: 1
DRWN 9 Drawing Page(s)
LN.CNT 1977
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method of preparing a sample of muscle tissue and of assaying the prepared sample to determine the presence of ***prions*** in the

sample is disclosed. The muscle tissue is homogenized and mixed with a complexing agent which forms a complex with a higher specific gravity than PrP.sup.Sc, the complexing agent or other components of the homogenate. Gravity is then used (e.g. ultra centrifugation) to concentrate the complex and the concentrate is assayed to detect ***prions*** . The muscle tissue is preferably extracted from a muscle or group of muscles such as hind limb muscle which have a higher or more preferably the highest concentration of ***prions*** as compared to other muscle in the mammal.

L1 ANSWER 7 OF 20 USPATFULL on STN
AN 2003:173177 USPATFULL
TI Capture compounds, collections thereof and methods for analyzing the proteome and complex compositions
IN Koster, Hubert, La Jolla, CA, UNITED STATES
Siddiqi, Suhaib, Oceanside, CA, UNITED STATES
Little, Daniel P., Winchester, MA, UNITED STATES
PI US 2003119021 A1 20030626
AI US 2002-197954 A1 20020716 (10)
PRAI US 2001-306019P 20010716 (60)
US 2001-314123P 20010821 (60)
US 2002-363433P 20020311 (60)
DT Utility
FS APPLICATION
LREP STEPHANIE SEIDMAN, HELLER EHRMAN WHITE & MCAULIFFE LLP, 7th FL., 4350 LA JOLLA VILLAGE DRIVE, SAN DIEGO, CA, 92122-1246
CLMN Number of Claims: 125
ECL Exemplary Claim: 1
DRWN 70 Drawing Page(s)
LN.CNT 6373
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Capture compounds and collections thereof and methods using the compounds for the analysis of biomolecules are provided. In particular, collections, compounds and methods are provided for analyzing complex protein mixtures, such as the proteome. The compounds are multifunctional reagents that provide for the separation and isolation of complex protein mixtures. Automated systems for performing the methods also provided.

L1 ANSWER 8 OF 20 USPATFULL on STN
AN 2003:64747 USPATFULL
TI Method for detecting ***prion*** proteins in tissue samples
IN Aslamkhan, Abubakr, Durham, NC, UNITED STATES
Higgins, Donald, Franklinton, NC, UNITED STATES
PI US 2003044868 A1 20030306
AI US 2001-924812 A1 20010808 (9)
DT Utility
FS APPLICATION
LREP PARADIGM GENETICS, INC, 108 ALEXANDER DRIVE, P O BOX 14528, RTP, NC, 27709-4528
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN 4 Drawing Page(s)
LN.CNT 778
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Surprisingly, the present inventors have discovered that thermal denaturation of ***prion*** protein facilitates its detection by immunological methods. Accordingly, the present invention provides methods for the preparation and thermal denaturation of samples for ***prion*** detection, comprising: homogenizing a candidate sample and heating said sample in a buffer, preferably one with properties that aid stabilization of the denatured form of the protein. The methods described in this disclosure can be used in the detection of PrP.sup.Sc. Such detection is useful for the diagnosis of transmissible spongiform encephalopathies. This method can be used with immunoassays of various formats, including, but not limited to, dot blot and western blot assays, which utilize polyclonal antibodies, monoclonal antibodies, antibody fragments, receptors, natural and synthetic ligands and other entities.

L1 ANSWER 9 OF 20 USPATFULL on STN

AN 2003:30296 USPATFULL
TI Protein aggregation assays and uses thereof
IN Kondejewski, Les, St. Lazare, CANADA
Chakrabartty, Avijit, Vaughan, CANADA
Qi, Xiao-Fei, Toronto, CANADA
Cashman, Neil, Toronto, CANADA
PI US 2003022243 A1 20030130
AI US 2002-176809 A1 20020620 (10)
PRAI US 2001-299849P 20010620 (60)
DT Utility
FS APPLICATION
LREP CLARK & ELBING LLP, 101 FEDERAL STREET, BOSTON, MA, 02110
CLMN Number of Claims: 115
ECL Exemplary Claim: 1
DRWN 23 Drawing Page(s)
LN.CNT 2602
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention features methods for identifying agents that modulate protein aggregation or stabilize protein conformation. Exemplary methods include an in vitro aggregation assay, a native state stabilization assay, a cell-based screening assay, and an animal-based screening assay. These methods can be used to identify agents useful for the treatment of conformational diseases resulting from aggregation of a protein.

L1 ANSWER 10 OF 20 USPATFULL on STN
AN 2003:17028 USPATFULL
TI Polymer conjugates of proteinases
IN Sherman, Merry R., San Carlos, CA, UNITED STATES
Martinez, Alexa L., San Jose, CA, UNITED STATES
Bhaskaran, Shyam S., San Bruno, CA, UNITED STATES
Williams, L. David, Fremont, CA, UNITED STATES
Saifer, Mark G., San Carlos, CA, UNITED STATES
French, John A., Santa Cruz, CA, UNITED STATES
PI US 2003012777 A1 20030116
AI US 2002-183607 A1 20020628 (10)
RLI Continuation-in-part of Ser. No. US 2002-103128, filed on 22 Mar 2002, PENDING Continuation-in-part of Ser. No. US 2001-894071, filed on 28 Jun 2001, ABANDONED
DT Utility
FS APPLICATION
LREP STERNE, KESSLER, GOLDSTEIN & FOX PLLC, 1100 NEW YORK AVENUE, N.W., SUITE 600, WASHINGTON, DC, 20005-3934
CLMN Number of Claims: 143
ECL Exemplary Claim: 1
DRWN 18 Drawing Page(s)
LN.CNT 2195
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods are provided for the stabilization of proteinases by the covalent attachment of or admixture with water-soluble polymers. The resultant stabilized proteinases have increased stability under the harsh conditions used in industrial genomics, which permits their use in the extraction and isolation of nucleic acids and the identification of disease-related ***prion*** proteins at elevated temperatures in solutions containing chaotropic agents, such as sodium dodecyl sulfate, urea or guanidinium salts, conferring advantages for robotic applications.

L1 ANSWER 11 OF 20 USPATFULL on STN
AN 2003:4268 USPATFULL
TI Sodium dodecyl sulfate compositions for inactivating ***prions***
IN Prusiner, Stanley B., San Francisco, CA, UNITED STATES
Supattapone, Surachai, Hanover, NH, UNITED STATES
PI US 2003004312 A1 20030102
US 6720355 B2 20040413
AI US 2002-56222 A1 20020122 (10)
RLI Continuation-in-part of Ser. No. US 2001-904178, filed on 11 Jul 2001, PENDING Continuation-in-part of Ser. No. US 2000-699284, filed on 26 Oct 2000, PENDING Continuation-in-part of Ser. No. US 2000-494814, filed on 31 Jan 2000, GRANTED, Pat. No. US 6322802 Continuation-in-part of Ser. No. US 1999-447456, filed on 22 Nov 1999, GRANTED, Pat. No. US 6331296

Continuation-in-part of Ser. No. US 1999-322903, filed on 1 Jun 1999, GRANTED, Pat. No. US 6214366 Continuation-in-part of Ser. No. US 1999-235372, filed on 20 Jan 1999, GRANTED, Pat. No. US 6221614 Continuation-in-part of Ser. No. US 1998-151057, filed on 10 Sep 1998, ABANDONED Continuation-in-part of Ser. No. US 1998-26957, filed on 20 Feb 1998, ABANDONED Continuation-in-part of Ser. No. US 1997-804536, filed on 21 Feb 1997, GRANTED, Pat. No. US 5891641
DT Utility
FS APPLICATION
LREP BOZICEVIC, FIELD & FRANCIS LLP, 200 MIDDLEFIELD RD, SUITE 200, MENLO PARK, CA, 94025
CLMN Number of Claims: 38
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 3471
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An antiseptic composition useful in destroying the infectivity of infectious proteins such as ***prions*** is disclosed. The antiseptic composition is preferably maintained at either a low pH of 4.0 or less or a high pH of 10.0 or more either of which allows for an environment under which the active component (which is preferably sodium dodecyl sulfate) destroys infectivity. The composition may be added to blood, blood products, collagen, tissues and organs prior to transplantation. The composition also may be added to livestock feed to denature any ***prions*** in the livestock. Methods of denaturing infectious proteins are also disclosed which method can use but do not require higher temperatures and long period of exposure.

L1 ANSWER 12 OF 20 USPATFULL on STN
AN 2002:258862 USPATFULL
TI Human endosulfine gene
IN Roch, Jean-Marc, Waukegan, IL, UNITED STATES
Scott, Victoria E.S., Evanston, IL, UNITED STATES
Anderson, Kristi L., Grayslake, IL, UNITED STATES
Sullivan, James P., Deerfield, IL, UNITED STATES
PI US 2002142432 A1 20021003
AI US 2001-824178 A1 20010402 (9)
RLI Continuation of Ser. No. US 1997-779775, filed on 7 Jan 1997, ABANDONED
DT Utility
FS APPLICATION
LREP Steven F. Weinstock, Abbott Laboratories, Department 377 / AP6D-2, 100 Abbott Park Road, Abbott Park, IL, 60064-6050
CLMN Number of Claims: 40
ECL Exemplary Claim: 1
DRWN 8 Drawing Page(s)
LN.CNT 2951
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides an isolated or purified polynucleotide that encodes human endosulfine polypeptide. Isoforms of human endosulfine are also disclosed. The invention also provides methods of making recombinant human endosulfine using the polynucleotides and host cells transformed with the polynucleotides.

L1 ANSWER 13 OF 20 USPATFULL on STN
AN 2002:246898 USPATFULL
TI Transgenic mice expressing human APP and TGF-.beta. demonstrate cerebrovascular amyloid deposits
IN Mucke, Lennart, Foster City, CA, United States
Wyss-Coray, Tony, Berkeley, CA, United States
Masliah, Eliezer, Chula Vista, CA, United States
PA The Regents of the University of California, Oakland, CA, United States (U.S. corporation)
PI US 6455757 B1 20020924
AI US 1999-262519 19990304 (9)
RLI Continuation-in-part of Ser. No. US 1997-947295, filed on 8 Oct 1997
DT Utility
FS GRANTED
EXNAM Primary Examiner: Crouch, Deborah
LREP Francis, Carol L., Borden, Paula A., Bozicevic, Field & Francis, LLP
CLMN Number of Claims: 14
ECL Exemplary Claim: 1

DRWN 9 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 1966
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention features non-human transgenic animal models for Alzheimer's disease (AD) and CAA, wherein the transgenic animal is characterized by 1) expression of bioactive transforming growth factor-.beta.1 (TGF-.beta.1) or 2) both expression of bioactive TGF-.beta.1 and expression of a human amyloid .beta. precursor protein (APP) gene product. The transgenic animals may be either homozygous or heterozygous for these alterations. Bigenic animals are further characterized by development of AD-associated and/or CAA-associated pathology within about two to three months of age, and at about twelve months of age are characterized by a reduced number of neuritic plaques relative to singly transgenic animals. The invention also features methods of screening for biologically active agents that facilitate reduction of .beta.-amyloid deposits in vivo and methods for facilitating reduction of formation of neuritic plaques in a subject susceptible to AD.

L1 ANSWER 14 OF 20 USPATFULL on STN
AN 2002:152685 USPATFULL
TI Compositions and methods for advanced glycosylation endproduct-mediated modulation of amyloidosis
IN Vitek, Michael P., 205 Park Knoll La., Apex, NC, United States 27502
Cerami, Anthony, Ram Island Dr., Shelter Island, NY, United States 11964
Bucala, Richard J., 504 E. 63rd St. Apt. 33-0, New York, NY, United States 10021
Ulrich, Peter C., 148 DeWolf Rd., Old Tappan, NJ, United States 07675
Vlassara, Helen, Ram Island Dr., Shelter Island, NY, United States 11964
Zhang, Xini, 150 Fairhaven Dr. Apt. D1, Jericho, NY, United States 117534)
PI US 6410598 B1 20020625
AI US 1995-477364 19950607 (8)
RLI Continuation-in-part of Ser. No. US 1995-457169, filed on 1 Jun 1995
Continuation-in-part of Ser. No. WO 1995-US1380, filed on 2 Feb 1995
Continuation-in-part of Ser. No. US 1994-311768, filed on 23 Sep 1994, now abandoned Continuation of Ser. No. US 1994-191579, filed on 3 Feb 1994, now abandoned
DT Utility
FS GRANTED
EXNAM Primary Examiner: Duffy, Patricia A.
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 12 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 2202
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates generally to the non-enzymatic glycosylation of amyloidogenic proteins and the consequent formation of advanced glycosylation endproducts (AGEs). It has been found that formation of AGE-amyloidogenic proteins can enhance amyloidosis. The invention further relates to compositions and methods for the prevention and treatment of amyloidosis associated with amyloid diseases, particularly neurodegenerative disease and Type II diabetes, and more particularly Alzheimer's disease. In a specific example, aggregation of an amyloidogenic peptide, .beta.AP, is enhanced by the glycosylation reaction of .beta.AP to form AGE-.beta.AP as defined herein. Accordingly, the invention extends to a method for modulating the in vivo aggregation of amyloid polypeptides and associated amyloidosis by controlling the formation and presence of AGE-amyloid polypeptide. A corresponding diagnostic utility comprises the measurement of the course and extent of amyloidosis by a measurement of the presence and amount of AGEs and particularly, AGE-amyloid. An assay is included that may use the AGE-amyloid polypeptide of the present invention to identify disease states characterized by the presence of AGE-amyloid. Additionally, such an assay can be utilized to monitor therapy and thus adjust a dosage regimen for a given disease state characterized by the presence of AGE-amyloid.

L1 ANSWER 15 OF 20 USPATFULL on STN

AN 2002:78206 USPATFULL
TI Antiseptic compositions for inactivating ***prions***
IN Prusiner, Stanley B., San Francisco, CA, UNITED STATES
Supattapone, Surachai, Hanover, NH, UNITED STATES
PI US 2002041859 A1 20020411
US 6719988 B2 20040413
AI US 2001-904178 A1 20010711 (9)
RLI Continuation-in-part of Ser. No. US 2000-699284, filed on 26 Oct 2000, PENDING Continuation-in-part of Ser. No. US 2000-494814, filed on 31 Jan 2000, GRANTED, Pat. No. US 6322802 Continuation-in-part of Ser. No. US 1999-447456, filed on 22 Nov 1999, PENDING Continuation-in-part of Ser. No. US 1999-322903, filed on 1 Jun 1999, GRANTED, Pat. No. US 6214366 Continuation-in-part of Ser. No. US 1999-235372, filed on 20 Jan 1999, GRANTED, Pat. No. US 6221614 Continuation-in-part of Ser. No. US 1998-151057, filed on 10 Sep 1998, ABANDONED Continuation-in-part of Ser. No. US 1998-26957, filed on 20 Feb 1998, ABANDONED Continuation-in-part of Ser. No. US 1997-804536, filed on 21 Feb 1997, GRANTED, Pat. No. US 5891641
DT Utility
FS APPLICATION
LREP Karl Bozicevic, Bozicevic, Field and Francis LLP, Suite 200, 200 Middlefield Road, Menlo Park, CA, 94025
CLMN Number of Claims: 22
ECL Exemplary Claim: 1
DRWN 12 Drawing Page(s)
LN.CNT 3354
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB An antiseptic composition useful in destroying the infectivity of infectious proteins such as ***prions*** is disclosed. The antiseptic composition is preferably maintained at a pH of 4.0 or less which allows for an environment under which the active component destroys infectivity. The composition may be added to blood, blood products, collagen, tissues and organs prior to transplantation. The composition also may be added to livestock feed to denature any ***prions*** in the livestock. Methods of denaturing infectious proteins are also disclosed.

L1 ANSWER 16 OF 20 USPATFULL on STN
AN 2002:37505 USPATFULL
TI METHOD OF PREPARING A STANDARD DIAGNOSTIC GENE TRANSCRIPT PATTERN
IN SHARMA, PRAVEEN, OSLO, NORWAY
LONNEBORG, ANDERS, AAS, NORWAY
PI US 2002022222 A1 20020221
US 6720138 B2 20040413
AI US 1999-429003 A1 19991029 (9)
RLI Continuation of Ser. No. WO 1998-GB1261, filed on 30 Apr 1998, UNKNOWN
PRAI NO 1997-2006 19970430
DT Utility
FS APPLICATION
LREP SUGHRUE MION ZINN MACPEAK & SEAS PLLC, 2100 PENNSYLVANIA AVENUE NW, WASHINGTON, DC, 200373213
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN 3 Drawing Page(s)
LN.CNT 1238
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for preparing a gene transcript pattern probe kit characteristic of a disease or condition or a stage thereof in a prokaryotic or eukaryotic organism using mRNA which is differentially expressed in the disease or condition or stage as probes, methods of diagnosis using the method and kits for performing the same are disclosed.

L1 ANSWER 17 OF 20 USPATFULL on STN
AN 2001:90277 USPATFULL
TI METHODS FOR IN VITRO SUSCEPTIBILITY TESTING OF CHLAMYDIA
IN STRATTON, CHARLES W, NASHVILLE, TN, United States
MITCHELL, WILLIAM M, NASHVILLE, TN, United States
PI US 2001002421 A1 20010531
US 6258532 B2 20010710
AI US 1998-25176 A1 19980218 (9)

RLI Continuation-in-part of Ser. No. US 1997-911593, filed on 14 Aug 1997, ABANDONED
DT Utility
FS APPLICATION
LREP KAREN F. ELBING, CLARK AND ELBING, 176 FEDERAL STREET, BOSTON, MA, 02110
CLMN Number of Claims: 55
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 763
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods for determining the susceptibility of intracellular pathogens, particularly Chlamydia, to single or combination of test agents are described. The methods can be used for in vitro or in vivo evaluation of agents that can be used as therapeutic agents in the treatment/eradication of pathogen infection in general or to target a specific infected organ. Assays which utilize nucleic amplification techniques (e.g., PCR) to determine effectiveness of the agent(s) evaluated are also described.

L1 ANSWER 18 OF 20 USPATFULL on STN
AN 2001:8223 USPATFULL
TI Transgenic mouse model of alzheimer's disease and cerebral amyloid angiopathy
IN Mucke, Lennart, Foster City, CA, United States
Wyss-Coray, Tony, Berkeley, CA, United States
Masliah, Eliezer, Chula Vista, CA, United States
PA The Regents of the University of California, Oakland, CA, United States (U.S. corporation)
PI US 6175057 B1 20010116
AI US 1997-947295 19971008 (8)
DT Utility
FS Granted
EXNAM Primary Examiner: Crouch, Deborah
LREP Francis, Carol L., Borden, Paula A.Bozicevic, Field & Francis LLP
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 1697
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention features non-human transgenic animal models for Alzheimer's disease (AD) and CAA, wherein the transgenic animal is characterized by 1) overexpression of bioactive transforming growth factor-.beta.1 (TGF-.beta.1) or 2) both overexpression of bioactive TGF-.beta.1 and expression of a human amyloid .beta. precursor protein (APP) gene product. The transgenic animals may be either homozygous or heterozygous for these alterations. Bigenic animals are further characterized by development of AD-associated and/or CAA-associated pathology within about two to three months of age.

L1 ANSWER 19 OF 20 USPATFULL on STN
AN 1999:92643 USPATFULL
TI Compositions and methods for stimulating amyloid removal in amyloidogenic diseases using advanced glycosylation endproducts
IN Vitek, Michael P., East Norwich, NY, United States
Cerami, Anthony, Shelter Island, NY, United States
Bucala, Richard J., New York, NY, United States
Ulrich, Peter C., Old Tappan, NJ, United States
Vlassara, Helen, Shelter Island, NJ, United States
Zhang, Xini, Jericho, NJ, United States
PA The Picower Institute For Medical Research, Manhasset, NY, United States (U.S. corporation)
PI US 5935927 19990810
WO 9520979 19950810
AI US 1996-501127 19960810 (8)
WO 1995-US1380 19950202
19960810 PCT 371 date
19960810 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 1994-311768, filed on 23 Sep 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-191579, filed on 3 Feb 1994, now abandoned
DT Utility

FS Granted
EXNAM Primary Examiner: Duffy, Patricia A.
LREP Klauber & Jackson
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 12 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 2154
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates generally to methods and compositions for treating amyloidogenic diseases such as Alzheimer's disease and the development of type II diabetes, in which deposition of amyloid in organs such as the brain and pancreas interfere with neurological function and insulin release, respectively. The methods and compositions are directed toward increasing the activity of scavenger cells within the body at recognizing and removing amyloid deposits from affected tissues and organs. Scavenger cells may be targeted to amyloid deposits by means of spontaneously-occurring chemical modifications called advanced glycosylation endproducts (AGEs). Compositions are described which increase scavenger cell activity towards AGE-modified amyloid. Amyloid removal may also be enhanced by increasing AGE levels in amyloid deposits within the body by administering AGE-modified amyloid targeting agents, which after becoming situated at sites containing amyloid, subsequently attract scavenger cells to degrade attendant amyloid. These methods and associated compositions result in a decrease in the extent of amyloid deposits in tissues, reducing the attendant pathology.

L1 ANSWER 20 OF 20 USPATFULL on STN
AN 94:11236 USPATFULL
TI Method of treating the symptoms of Alzheimer's disease
IN Wagle, Sudhakar S., Mequon, WI, United States
Steinbach, Thomas, Houston, TX, United States
Lawyer, Carl H., Mequon, WI, United States
Hermann, William J., Sealy, TX, United States
Gawish, Ali A. S., Mequon, WI, United States
PA Kremers-Urban Company, Mequon, WI, United States (U.S. corporation)
PI US 5284664 19940208
AI US 1992-835029 19920205 (7)
RLI Continuation-in-part of Ser. No. US 1991-803844, filed on 4 Dec 1991 which is a continuation-in-part of Ser. No. US 1991-728267, filed on 11 Jul 1991, now abandoned which is a continuation of Ser. No. US 1988-228364, filed on 4 Aug 1988, now patented, Pat. No. US 5055296
DT Utility
FS Granted
EXNAM Primary Examiner: Robinson, Douglas W.; Assistant Examiner: Witz, Jean C.
LREP Tilton, Fallon, Lungmus & Chestnut
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 729
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A therapeutic method for treating Alzheimer's or related disease. The method comprises administering a therapeutically-effective amount of a mammalian liver extract, the extract being characterized by being heat stable, insoluble in acetone and soluble in water, peptide or peptide fragment selected from the groups consisting of Sequence Identification Numbers 1-9.